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## Examination of multiple fatigue models during prolonged cycling in hot versus cold climates

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EDITH COWAN UNIVERSITY

School of Biomedical and Sports Science

**EXAMINATION OF MULTIPLE FATIGUE MODELS DURING PROLONGED  
CYCLING IN HOT VERSUS COLD CLIMATES**

HONOURS THESIS

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**Degree:** Bachelor of Science (Hons)

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**Date of Submission:**

## USE OF THESIS

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## ABSTRACT

Much of the previous research into understanding fatigue during prolonged cycling has found that endurance performance may be limited by numerous physiological, biomechanical, environmental, mechanical, and psychological factors. From the vast body of knowledge available, a number of differing models have been developed in order to better understand the specific mechanisms of fatigue during prolonged cycling. These models include: i) the cardiovascular/anaerobic model, ii) the energy supply/energy depletion model, iii) the neuromuscular fatigue model, iv) the muscle trauma model, v) the biomechanical model, vi) the thermoregulatory model, vii) the psychological/motivational model, and viii) the central governor model. From the literature presently available, fatigue would appear to be task- and condition-specific. Studies demonstrating this, however, are limited. The purpose of this thesis, therefore, was to examine variables from multiple fatigue models concurrently to determine which variables were best related to fatigue during prolonged endurance exercise in hot versus cold conditions. Following a 100-km familiarization time trial (22°C), nine endurance-trained male cyclists ( $\dot{V}O_{2\max} = 62.1 \pm 8.5 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) completed two 100-km time trials, interspersed with five 1-km sprints, and four 4-km sprints, in hot (34°C) and cold (10°C) conditions. Rectal and skin temperatures, muscle activation of the lower limb via integrated electromyography (iEMG), expired gases, as well as blood measures of lactate, glucose, urea, bicarbonate hematocrit, and the electrolytes  $\text{Na}^+$ ,  $\text{K}^+$ , and  $\text{Cl}^-$  were measured. Rectal temperature increased significantly in both the hot and cold trials ( $P < 0.001$ ) and was significantly lower in the cold trial at nearly all measurements beyond 28-km ( $P < 0.05$ ; with exception of 62- and 88-km). Power output was significantly lower in the hot trial (22-km;  $P < 0.05$ ) prior to marked differences in rectal temperature (42-km;  $P < 0.05$ ). iEMG of biceps femoris and soleus was also significantly lower in the hot trial (22-km and 15-km, respectively;  $P < 0.05$ ) prior to significant differences in rectal temperature. iEMG of vastus lateralis, however, was not significantly different between trials. Mean skin, mean body temperature and perceived thermal sensation were greater in the hot trial compared to the cold (all  $P < 0.001$ ). Blood

sodium concentration was significantly lower in the hot trial compared to the cold ( $P<0.05$ ). Perceived exertion, pain intensity in the quadriceps, heart rate, blood lactate, pH, and glucose concentration were not statistically different between trials. In the cold trial, power output declined during the 1-km and 4-km sprints ( $P<0.05$ ), while iEMG of vastus lateralis and biceps femoris decreased significantly during 4-km sprints ( $P<0.05$ ). Thus, reductions in iEMG and power output during exercise in the heat may be a centrally controlled anticipatory response to increases in mean skin, mean body, and thermal sensation; the reduced power output would lead to a reduction in heat production ensuring that the task can be completed without the development of hyperthermia. In the alternative cold condition, reductions in power output and iEMG occurred despite significantly lower mean skin, mean body and rectal temperature, suggesting that central reductions in motor drive in the cold are unrelated to thermal stress. Possible reasons for the reduced sprint power output in the cold are uncertain, but may be related to a reduction in muscle glycogen stores or perhaps a limited oxygen supply to the active muscles.



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# **CHAPTER 1      INTRODUCTION**

## **1.1      Background**

Road cycling is a complex sport, whereby in each professional cycling season there are 90 to 100 competition days, comprising of one-day races, one-week tour races, and three-week tour races (Lucia, Hoyos, & Chicharro, 2001; Lucia, Hoyos, Santalla, Earnest, & Chicharro, 2003). Within each of these races, participants may perform a number of competition requirements: flat, long stages, individual time trials and uphill ascents (de Koning, Bobbert, & Foster, 1999; Lucia et al., 2001), requiring individual and team strategic approaches (de Koning et al., 1999; Nikolopoulos, Arkinstall, & Hawley, 2001). Of each of these requirements, the individual time trial is considered vital to the overall standings of a race (Lucia et al., 2001; Padilla, Mujika, Orbananos, & Angulo, 2000). The time trial racing format is of a 'closed-loop design', whereby cyclists individually ride a known given distance in the shortest possible time (Padilla et al., 2000). In this type of race, or component of a race, various physiological systems are pushed to exhaustion (de Koning et al., 1999), making pacing by regulation of speed essential to completion of the event (de Koning et al., 1999).

To date, a vast number of investigations have examined many physiological ( $\dot{V}O_{2\max}$ , anaerobic threshold, economy and efficiency of movement) (Balmer, Davison, & Bird, 2000; Jeukendrup, Craig, & Hawley, 2000; Laursen, Shing, & Jenkins, 2003a; Lucia, Hoyos, Carvajal, & Chicharro, 1999; Lucia, Joyos, & Chicharro, 2000), environmental (wind, temperature, altitude and humidity) (Jeukendrup et al., 2000; Kay et al., 2001), and biomechanical (positioning) (de Koning et al., 1999; Garside & Doran, 2000; Jeukendrup et al., 2000) factors relating to time trial performance. During prolonged time-trials, all of these variables play a significant role in the eventual outcome, with the most important being maximal maintainable power and reductions in aerodynamic drag (Candau et al., 1999; de Koning et al., 1999; Jeukendrup et al., 2000).

The ability to maintain a high power output during prolonged cycling is limited by the ability of the cyclist to resist fatigue. What precisely causes this fatigue, however, is controversial. Indeed, understanding fatigue during prolonged endurance exercise has been a major research agenda of Exercise Scientists for more than 80 years (Hill, Long, & Lupton, 1924). Still however, despite numerous theories and models, no precise explanation exists (Noakes, 2000b). The physiological, biochemical, biomechanical, and cognitive models used to explain fatigue are diverse (Brooks, Fahey, White, & Baldwin, 2000b; Hampson, St Clair Gibson, Lambert, & Noakes, 2001; Hunter, St Clair Gibson, Lambert, Nobbs, & Noakes, 2003; Noakes, 2000b). This may be due to the fact that precise fatigue mechanisms will vary according to the specifics of the task. For example, Tordi et al. (2003) recently showed, during two bouts of repeated six-minute cycling exercise ( $>85\% \dot{V}O_{2max}$ ), that performance was limited by the supply of sufficient oxygen to the working muscles. However, Kay et al. (2001) have recently showed that during prolonged cycling (60-min self-paced time trial), decrements in power production were the result of central and peripheral neuromuscular alterations. More recently, Tucker et al. (2004) proposed that fatigue during a 20-km cycle time trial in a hot (35°C) versus a cool (15°C) climate was the result of a “teleoanticipatory” reduction in muscle activation aimed at limiting the rate and overall rise in core temperature. However, as a limited number of variables were recorded in each of these studies, it is difficult to ascertain whether or not other factors (i.e., other fatigue models) were responsible for the observed reductions in power output. Indeed, the fatigue process appears to be task specific (Hunter, Duchateau, & Enoka, 2004). Due to the dynamic and irregular nature of endurance cycling coupled with the varying environmental conditions in which competitions take place, consequent fatigue-related reductions in power production appear to be multifactorial. Nevertheless, very few studies have attempted to examine multiple fatigue models concurrently (Hunter, St Clair Gibson, Mbambo, Lambert, & Noakes, 2002; Tucker et al., 2004).

Understanding mechanisms of fatigue during exercise is an important initiative for Exercise Scientists if we are to further contribute to the performances of elite endurance athletes. If factors determining fatigue and athletic performance can be established more definitively, it will help us to determine which training adaptations are

most important for increasing exercise performance, or how training should be structured to maximise those adaptations (Noakes, 2000b; Noakes, Peltonen, & Rusko, 2001).

## **1.2 Purpose of the Study**

The purpose of this research was to develop a better understanding of the fatigue process during prolonged endurance cycling. The present study examined the relationship between numerous physiological (core body temperature, skin temperature, oxygen consumption, heart rate), biochemical (capillary blood variables), biomechanical (muscle activation of the lower limb), cognitive (perceived exertion, pain and thermal sensation) variables and decrements in cycling power output in well-trained cyclists during prolonged and dynamic (interspersed sprints) endurance cycling in hot (34°C) and cold (10°C) climates.

## **1.3 Significance of the Study**

Despite a vast body of available knowledge that has examined specific aspects of fatigue, very little research has examined multiple aspects of fatigue concurrently. A detailed assessment of key determinants of fatigue during such an event will provide further insight into the factors leading to reduced performance and fatigue during prolonged endurance exercise. To date, it is uncertain whether an alteration of task-specific intensity and/or climatic conditions has any bearing on the nature of the fatigue process. This study differs to other similar studies that have concurrently examined numerous fatigue models (Tucker et al., 2004), as in the present study more variables were measured, subjects rode for a greater distance and numerous intensities were examined.

## **1.4 Research Questions**

- i. How do differences in ambient temperature affect core body temperature, muscle activation of the lower limb, biochemical markers in the blood, cardio-respiratory measures, psychological factors and decrements in cycling power output (i.e., fatigue)?
- ii. Are differences in cycling intensity power output during a prolonged ride related to core body temperature, muscle activation of the lower limb, biochemical markers in the blood, cardio-respiratory measures, and psychological factors?
- iii. What are the most strongly associated factors related to fatigue in the differing environmental conditions and differing cycling intensities?

## **1.5 Hypotheses**

- i. Cycling performance (i.e., power output or performance time) will be significantly lower in the hot when compared to the cold environmental condition. This will be associated with significant increases in core temperature and a consequent decrease in lower limb muscle activation.
- ii. Decreases in cycling intensity (i.e., decreased power output) will be associated with decreases in muscle activation of the lower limb, and increases in blood lactate, oxygen consumption, and core body temperature. No differences will be seen with perceived exertion between conditions.
- iii.
  - a) Decrements in cycling power output will be associated with significant rises in core temperature and “teleoanticipatory” reductions in muscle activation of the lower limb during cycling in a hot environment.
  - b) Decrements in cycling power output will be associated with significantly reduced levels of blood pH and blood glucose causing a reduction in muscle activation of the lower limb during exercise in the cold climate.

## **1.6 Limitations**

Although this study had a number of advantages over prior related research, some limitations did exist:

- i. It was assumed that the subjects tested in this study were indicative of the target population (i.e., professional cyclists). To regulate this limitation, only the best possible subjects from the greater Perth area were selected.
- ii. Subjects also performed the time trials under clinical laboratory conditions which were considered to be indicative of race conditions. However, for the purposes of this study, the environment (i.e., temperature and wind conditions) needs to be strictly controlled. Thus, a virtual racetrack was designed with the use of the Velotron software in order to simulate an actual time trial.
- iii. Subjects were not permitted to wear arm-warmers in the cold time trial condition, which is sometimes practiced in the field.
- iv. Water was consumed *ad libitum* during this study, which made the rates of fluid consumption higher in the hot condition. Nevertheless, this limitation was justified to prevent dehydration (Dennis, Noakes, & Hawley, 1997; Galloway, 1999).
- v. 34°C was deemed to be representative of a hot environment.
- vi. 10°C was deemed to be representative of a cold environment.

## **1.7 Delimitations**

Subjects were cyclists aged between 18 and 41 years, and the data arising from this study was representative of that group.

## **1.8 Definitions of Selected Terms**

<b>ATP:</b>	<b>Adenosine triphosphate</b>
<b>BDC:</b>	<b>Bottom dead centre</b>
<b>BF:</b>	<b>Biceps femoris</b>
<b>BPM:</b>	<b>Beats per minute</b>
<b>CHO:</b>	<b>Carbohydrate</b>
<b>EMG:</b>	<b>Electromyography</b>
<b>iEMG:</b>	<b>Integrated electromyography</b>
<b>IT/RT:</b>	<b>Inspection time / Reaction time</b>
<b>MFI-20:</b>	<b>Multidimensional fatigue inventory</b>
<b>MVIC:</b>	<b>Maximum voluntary isometric contraction</b>
<b>MPFS:</b>	<b>Median power frequency spectrum</b>
<b>PPO:</b>	<b>Peak power output</b>
<b>RER:</b>	<b>Respiratory exchange ratio</b>
<b>RPE:</b>	<b>Rate of perceived exertion</b>
<b>Sol.:</b>	<b>Soleus</b>
<b>TDC:</b>	<b>Top dead centre</b>
<b>VL:</b>	<b>Vastus lateralis</b>
<b><math>\dot{V}_E</math>:</b>	<b>Minute ventilation</b>
<b><math>\dot{V}O_2</math>:</b>	<b>Oxygen consumption</b>
<b><math>\dot{V}O_{2max}</math>:</b>	<b>Maximal oxygen consumption</b>

## **CHAPTER 2      REVIEW OF LITERATURE**

The following review of literature was submitted for publication in *Sports Medicine* on the 1<sup>st</sup> of July, 2004. At present time, this manuscript is still under review.

### **2.1      Defining Fatigue**

The word 'fatigue' is generally used to define sensations of tiredness and associated decrements in muscular performance and function (Brooks et al., 2000b; Green, 1997; Kay & Marino, 2000; Kay et al., 2001; Millet et al., 2000; Millet, Lepers et al., 2002; Pinniger, Steele, & Groeller, 2000; St Clair Gibson, Lambert, & Noakes, 2001; Wilmore & Costill, 1999). It is therefore important to note that exercise is terminated at exhaustion, and not a point of fatigue (Kay & Marino, 2000; Kay et al., 2001). Fatigue is considered to be a safety mechanism aimed at preventing injury or death occurring during exercise (Gabriel, Basford, & An, 2001; Kay & Marino, 2000; Noakes, 2000b; Pinniger et al., 2000). As fatigue is considered an inevitable and negative consequence of physical activity (Kay et al., 2001; Pinniger et al., 2000), there has been much research into its effect upon exercise performance (Kay, St Clair Gibson, Mitchell, Lambert, & Noakes, 2000; Lepers, Hausswirth, Maffiuletti, Brisswalter, & van Hoecke, 2000; Lepers, Maffiuletti, Rochette, Brugniaux, & Millet, 2002; Lepers, Millet, Maffiuletti, Hausswirth, & Brisswalter, 2001; Millet et al., 2000; Millet, Lepers et al., 2002; Millet, Millet, Lattier, Maffiuletti, & Candau, 2003; Paasuke, Ereline, & Gapeyeva, 1999; Rodacki, Fowler, & Bennett, 2001; St Clair Gibson, Lambert et al., 2001).

Extensive research into fatigue-induced decrements in exercise performance spans across a variety of sports science disciplines. This has caused a differentiation in the theories that explain the specific process (physiological, neurophysiological, biomechanical and/or psychological) responsible for decrements in exercise performance. As a result, numerous models have been developed to explain the fatigue

response that inevitably occurs during exercise. These include the cardiovascular/anaerobic model, the energy supply/energy depletion model, the neuromuscular fatigue model, the muscle trauma model, the biomechanical model, the thermoregulatory model, the psychological/motivational model, and the central governor model.

## **2.2 Cardiovascular/Anaerobic Model**

The cardiovascular/anaerobic model of fatigue states that fatigue occurs when the heart is no longer able to supply oxygen and remove waste products to and from the working muscles at the required rate (Ainsworth, Serfass, & Leon, 1993; Bilodeau, Henderson, Nolte, Pursley, & Sandfort, 2001; Brooks, Fahey, White, & Baldwin, 2000a; Delp & Laughlin, 1998; Green, 1997; Noakes, 2000b; Noakes et al., 2001). This model is supported by the observation that endurance training increases both cardiac output and the capacity of the muscles to utilise oxygen (Radegran, Blomstrand, & Saltin, 1999; Saltin, Radegran, Koskolou, & Roach, 1998). This ability can be quantified in terms of one's maximal oxygen uptake ( $\dot{V}O_{2max}$ ) (Brooks et al., 2000a; Gissane, Corrigan, & White, 1991; MacDougall, Wenger, & Green, 1991; Noakes, 2000b). Professional level endurance cyclists possess  $\dot{V}O_{2max}$  values of between 70 and 80 ml·kg<sup>-1</sup>·min<sup>-1</sup>, or 5.0-5.5 L·min<sup>-1</sup> (Jeukendrup et al., 2000; Lucia, Hoyos, Margarita, Santalla, & Chicharro, 2002; Lucia, Hoyos, Santalla, Perez, & Chicharro, 2002). These high levels of  $\dot{V}O_{2max}$  are achieved through increases in oxygen delivery and utilisation at the tissue level (Faulkner, Heigenhauser, & Schork, 1977; Tonkonogi & Sahlin, 1997). Oxygen delivery can be modified by changes in cardiac output (Delp & Laughlin, 1998; Faulkner et al., 1977; Hahn & Gore, 2001), red blood cell mass, (Hahn & Gore, 2001) and potentially plasma volume (Warburton, Gledhill, Jamnik, Krip, & Card, 1999).



## **2.2.1 Oxygen delivery**

### **2.2.1.1 Cardiac output**

Cardiac output is increased via an increase in stroke volume and heart rate (Brooks et al., 2000a; de Vries & Housh, 1994; Powers & Howley, 1997; Wilmore & Costill, 1999). There is, however, a limit to the capacity of the heart to pump blood to the working muscles (Delp & Laughlin, 1998; Radegran et al., 1999). The specific adaptations to endurance training have been well documented whereby cardiac output can be significantly increased following endurance training (Saltin et al., 1998). Both highly-trained and elite cyclists possess greater left ventricular dilation and hypertrophy than untrained individuals, thus permitting a greater stroke volume during exercise (Hoogsteen, Hoogeveen, Schaffers, Wijn, & van der Wall, 2003). These adaptations to training insinuate that performance during cycling exercise may be limited by an insufficient supply of oxygen to the working muscles via cardiac output (Brooks et al., 2000a, 2000b; Green, 1997; Tonkonogi & Sahlin, 1997). Support for this model has been shown by González-Alonso and Calbet (Gonzalez-Alonso & Calbet, 2003), who examined cardiac output, mean arterial pressure, muscle blood flow and muscle  $O_2$  delivery during exhaustive maximal aerobic cycling (356 W) in trained subjects. It was found that under both thermally elevated (i.e., skin temperature  $10^{\circ}\text{C}$  and core body temperature  $1^{\circ}\text{C}$  above normally levels) and thermally neutral conditions there is a limited cardiac output and mean arterial pressure which leads to limited muscle blood flow,  $O_2$  delivery, and  $O_2$  uptake (Gonzalez-Alonso & Calbet, 2003). Under heat stress there was also a reduced  $\dot{V}O_{2\max}$  caused by an earlier limitation in cardiac output and mean arterial pressure (Gonzalez-Alonso & Calbet, 2003). It was concluded that as leg arterial-venous  $O_2$  difference and extraction progressively increased till the end of the test and not reach a maximum (91%) then limitations in the diffusion of oxygen at the muscle did not cause fatigue (Gonzalez-Alonso & Calbet, 2003). Interestingly, Wolfel et al. (1998) examined the effects of a  $\beta$ -adrenergic blockade (80 mg of propranolol every four hours) on submaximal cycling ( $50\% \dot{V}O_{2\text{peak}}$ ) at sea level and at altitude (4,300 m) in endurance-trained individuals. Propranolol was found to successfully reduce cardiac output at sea level, on arrival, and following 21 days at altitude, compared to the placebo

group. This occurred despite a greater stroke volume at altitude (i.e., reduced heart rate). This effectively reduced systemic O<sub>2</sub> delivery (blood flow x arterial O<sub>2</sub> content) in the propranolol group and caused greater compensatory systemic O<sub>2</sub> extraction (determined via femoral arterial and venous catheterisation) (Wolfel et al., 1998). As whole body  $\dot{V}O_2$  was not significantly different between the two groups, it was suggested that reductions in cardiac output were initially offset by an increased O<sub>2</sub> extraction by the muscle (Wolfel et al., 1998). Therefore, further studies need to be conducted to determine whether or not cardiac output or perhaps O<sub>2</sub> extraction (see oxygen utilisation) limits cycling performance at exercise intensities similar to those achieved by professional endurance cyclists.

#### **2.2.1.2 Red blood cell mass and plasma volume**

The performance enhancing effect of an increase in blood volume and/or red blood cell mass on the delivery of oxygen to the working muscles has been well documented (Hahn & Gore, 2001; Levine & Stray-Gundersen, 1997; Warburton et al., 1999). Elite endurance trained cyclists possess a high blood volume (6648 mL, 95 mL.kg<sup>-1</sup>) (Warburton et al., 1999) compared to untrained individuals (4876 mL) (Coyle, Hopper, & Coggan, 1990). Artificial increases in plasma volume, irrespective of maintained haemoglobin [Hb] content, should lead to a reduction in the content of oxygen in the blood, thus causing a decline in maximal exercise performance (Warburton et al., 1999). On the other hand, Warburton et al. (1999) showed that an artificial plasma volume expansion of approximately 8% (500 mL infusion of 6% dextran) in elite cyclists had no effect on  $\dot{V}O_{2max}$ . Plasma volume expansion did, however, lead to an increase in cardiac output and stroke volume signifying that performance at  $\dot{V}O_{2max}$  is not limited by the ability of the heart to pump blood (cardiac output), but perhaps by a combination of blood volume and haemoglobin content (Warburton et al., 1999).

If exercise performance is indeed limited by the supply of oxygen rich blood to the muscles, then increasing the oxygen carrying capacity of the blood should cause significant improvements. Much of the work done in this area has examined the effects

of red blood cell reinfusion (blood doping), erythropoietin (EPO) (Hahn & Gore, 2001) or altitude acclimatisation (Hahn & Gore, 2001; Levine & Stray-Gundersen, 1997) on maximal and submaximal exercise performance. It has been well established that cycling performance can be improved via increasing the red blood cell concentration (Hahn & Gore, 2001; Warburton et al., 1999). This is supported by the fact that well-trained endurance cyclists tend to have higher levels of resting haemoglobin ( $15.3 \text{ g} \cdot 100 \text{ mL}^{-1}$ ) and hematocrit (45.1%) (Warburton et al., 1999) than male mountain climbers ( $13.8 \text{ g} \cdot 100 \text{ mL}^{-1}$  and 43.3%, respectively) (Robertson et al., 1982). The practice of EPO and/or blood doping has been alleged to be used by a number of elite cyclists in order to improve cycling performance, however this practice is dangerous as it increases the viscosity of the blood resulting in an increased likelihood of stroke, heart attack, heart failure and pulmonary edema (McArdle, Katch, & Katch, 2001). Therefore the International Cycling Union has set legal hematocrit, haemoglobin and reticulocyte levels of 50% (47% for females) (International\_Cycling\_Union, 2001; McArdle et al., 2001),  $17 \text{ g/dL}$  ( $10.5 \text{ mmol}$ ), and 2.4% ( $120,000 \text{ } \mu\text{L}$ ) (International\_Cycling\_Union, 2003), respectively. While individuals can naturally exceed this limit ( $\sim 3\%$  of humans), hematocrit levels in professional cyclists during the fourth and seventh rounds of the 2000 Tour de Suisse were reported on average to be 44.5% (International\_Cycling\_Union, 2001). The significant influence that endurance training has on the ability to supply oxygen to the working muscles provides evidence that exercise performance is limited by the variables inside the cardiovascular/anaerobic model (Figure 1) (Hahn & Gore, 2001). Further support for the cardiovascular/anaerobic model stems from studies that have examined the occlusion of blood flow to muscles, the utilisation of oxygen by the muscles, and the accumulation of metabolic byproducts.

### **2.2.1.3 Muscle blood flow occlusion**

Muscle blood flow plays a significant role in the delivery of substrates and is therefore thought to have a significant effect on the development of fatigue (Allman & Rice, 2002; Delp & Laughlin, 1998; Noakes, 2000b; Noakes et al., 2001). During steady state submaximal exercise, there tends to be a linear relationship between muscle blood flow and power output (Delp & Laughlin, 1998; Saltin et al., 1998) which is also related to

the active muscle mass (Radegran et al., 1999; Saltin et al., 1998), haemoglobin concentration of the blood, and the arterial oxygen content (Saltin et al., 1998). There is, however, a limited ability of the heart to supply blood to the working muscles. During dynamic knee extension exercise, large ranges in peak muscle perfusion ( $149\text{--}373\text{ ml}\cdot\text{min}^{-1}\cdot 100\text{ g muscle}^{-1}$ ) have been reported (Radegran et al., 1999), with cyclists reporting values as high as  $380\text{ ml}\cdot\text{min}^{-1}\cdot 100\text{ g muscle}^{-1}$  (Blomstrand, Radegran, & Saltin, 1997). During dynamic knee extensor exercise, Radegran et al. (1999) showed that muscle perfusion of the quadriceps femoris did not differ when ~64% of the muscle mass was active, compared to when 100% of the muscle was active. This occurred despite further increases in muscle oxygen uptake, suggesting a limit to muscle blood flow. Thus, increases in muscle  $\dot{V}O_2$  may be due to an increased oxygen extraction by the working muscle (Radegran et al., 1999).

Attenuation in muscle blood flow during cycling could be the result of blood flow occlusion (Allman & Rice, 2002). During long slow tetanic muscular contractions such as in low cadence cycling, greater blood flow occlusion occurs in the muscle, which could reduce muscle blood flow (Allman & Rice, 2002). Increases in blood flow are thought to be due to greater efficiency of the muscle to act as a pump, thereby facilitating venous blood flow return during high cadence cycling (Gotshall, Bauer, & Fahrner, 1996). Interestingly, it has been suggested that the age-related loss in muscle mass may actually reduce skeletal muscle blood flow occlusion and therefore improve the endurance of older individuals in specific sub-maximal tasks (Allman & Rice, 2002).

### **2.2.2 Oxygen utilisation**

It is well documented that endurance training increases the aerobic potential or oxidative power (oxidative phosphorylation rate) of muscle (Tonkonogi & Sahlin, 1997). Aerobically trained skeletal muscle contains greater mitochondrial density (size and volume) (Bizeau, Wills, & Hazel, 1998; Hood, Takahashi, Connor, & Freyssenet, 1999; Hoppeler & Fluck, 2003; McArdle et al., 2001; Tonkonogi, Walsh, Svensson, & Sahlin, 2000; Walsh, Tonkonogi, & Sahlin, 2001), aerobic enzyme activity (Bizeau et al., 1998; Hood et al., 1999; Hoppeler & Fluck, 2003; Paasuke et al., 1999; Pringle et al.,

2003; Tonkonogi et al., 2000; Walsh et al., 2001), capillarisation (Kraus, Stallings, Yeager, & Gavin, 2004; Pringle et al., 2003), and myoglobin (Hoppeler & Fluck, 2003; Paasuke et al., 1999). These increases allow a greater aerobic adenosine triphosphate (ATP) production by the mitochondria (Bizeau et al., 1998; Hood et al., 1999; McArdle et al., 2001; Walsh et al., 2001). This is supported by numerous studies that have found significant relationships to exist between mitochondrial density (Hahn & Gore, 2001; Kirkenall & Garrett, 1998; Tonkonogi & Sahlin, 2002; Tonkonogi et al., 2000), aerobic enzyme activity (Bizeau et al., 1998; Hahn & Gore, 2001; Pette, 1998; Tonkonogi & Sahlin, 2002; Tonkonogi et al., 2000), muscle capillarisation (Kirkenall & Garrett, 1998; Kraus et al., 2004; Pringle et al., 2003) and oxygen consumption. Bizeau et al. (1998) found that six weeks of endurance running in rats resulted in significant increases in the maximal  $\dot{V}O_2$  of subsarcolemmal (36%) and intermyofibrillar (20%) mitochondria. The authors suggested that such substantial increases in mitochondrial oxidative power were the result of increases in electron transport chain and matrix enzyme activity (Bizeau et al., 1998). The influence of endurance training on the oxidative power of humans was later examined by Tonkonogi et al. (2000), who found that after six weeks of endurance cycle training,  $\dot{V}O_{2\max}$  increased by 24% accompanied by a sharp increase in mitochondrial (citrate synthase) enzyme activity (21.1 - 31.0 mmol.min<sup>-1</sup>). After endurance training, however, no significant change was found between the ratio of maximal mitochondrial oxygen consumption to whole body  $\dot{V}O_{2\max}$ , suggesting that the efficiency of mitochondrial oxidative power remains unchanged after endurance training. In contrast, Walsh et al. (2001) found that after six weeks of endurance training (4 x 1-hour cycling sessions at 50%, 70% and 100% of  $\dot{V}O_{2\max}$ ) increases in muscle oxidative potential (38%) were greater than that of whole body  $\dot{V}O_{2\max}$  (24%). From this, it was viewed that  $\dot{V}O_{2\max}$  is closely associated with cardiac output whereas, peak muscle oxidative potential restricts prolonged endurance performance (Walsh et al., 2001). Hoppeler et al. (2003) suggested that the discrepancies that exist between increases in  $\dot{V}O_{2\max}$  and muscle oxidative potential are due to the fact that only a small volume of the muscle is specifically trained.

## 2.2.3 Metabolite accumulation

### 2.2.3.1 Lactic acid

During high intensity cycling exercise, lactic acid can accumulate due to an imbalance between its production and removal (Ainsworth et al., 1993; Bogdanis, Nevill, & Lakomy, 1994; Brooks et al., 2000b; Juel, 1998; Lucia et al., 2001; Lucia, Hoyos, Santalla et al., 2002; MacDougall et al., 1991). The generation of lactic acid in the muscles results in a lowering of the pH of the muscle and blood due to the dissociation of lactic acid into lactate and hydrogen ions ( $H^+$ ) (Bogdanis et al., 1994; Brooks et al., 2000b; Juel, 1998; Martini, 2001). During a maximal incremental cycling test performed by professional cyclists, lactate response in capillary blood at exhaustion ( $\dot{V}O_{2max}$ ) was found to be significantly higher in hill climbers ( $\sim 7.0 \text{ mmol}\cdot\text{L}^{-1}$ , 480 W) compared to time trialists ( $\sim 5.0 \text{ mmol}\cdot\text{L}^{-1}$ , 520 W) (Lucia et al., 2000). Stepto et al. (2001) showed significant increases in muscle lactate ( $6.2 \text{ mmol}\cdot\text{kg}^{-1}$  dry mass to  $32.7 \text{ mmol}\cdot\text{kg}^{-1}$  dry mass) and significant decreases in muscle pH (7.09 to 7.01) in highly-trained cyclists following eight repeated bouts (5-min duration) of high-intensity cycling (86%  $\dot{V}O_{2max}$ ). The reduced intramuscular pH may decrease glycolytic flux by inhibiting phosphofructokinase (PFK), interrupting contractions by reducing  $Ca^{2+}$  release and removing  $Ca^{2+}$  from troponin (Brooks et al., 2000b; Hill, Thompson, Ruell, Thom, & White, 2001; Stackhouse, Reisman, & Binder-Macleod, 2001), stimulating pain receptors (Brooks et al., 2000b; Hampson et al., 2001; Hill et al., 2001; Stackhouse, Reisman et al., 2001) and consequently diminishing performance (Davis & Bailey, 1997; Stackhouse, Reisman et al., 2001). Furthermore,  $H^+$  ions that are released into the blood may affect performance by influencing pain receptors in the brain (Bogdanis et al., 1994), inhibiting  $O_2$  transportation via haemoglobin (Brooks et al., 2000b), and reducing the dissociation of free fatty acids into the blood (Brooks et al., 2000b). Removal of  $H^+$  ions via an increased speed of blood flow therefore appears critical in the reduction of fatigue.

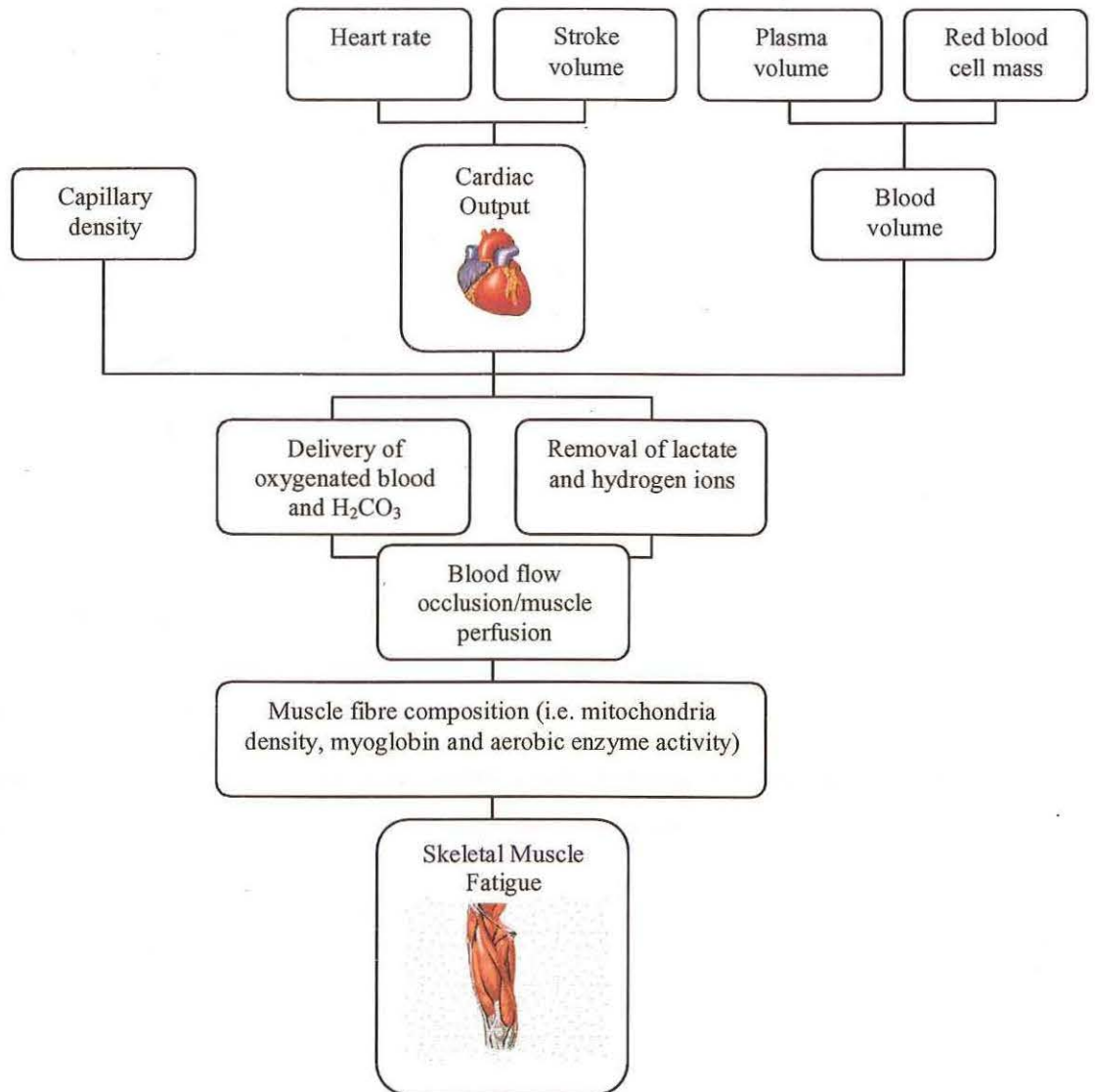
Well-trained cyclists tend not only to report high  $\dot{V}O_{2max}$  values (Jeukendrup et al., 2000; Lucia, Hoyos, Santalla et al., 2002), but their anaerobic threshold occurs at a

greater percentage of their  $\dot{V}O_{2\max}$  (Lucia et al., 2001), suggesting an increased fatigue resistance of type I fibres, and a greater ability to oxidise fat and reduce lactate accumulation at a given workload (Lucia, Pardo, Duranuez, Hoyos, & Chicharro, 1998). Studies examining lactate accumulation during cycling have found that a strong correlation exists between increases in lactate concentration and reductions in power output above threshold levels (Ainsworth et al., 1993; Liedl, Swain, & Branch, 1999). Due to high anaerobic and lactate thresholds (~90% of  $\dot{V}O_{2\max}$ ) and maximal power output (>500 W) (Lucia et al., 2001; Lucia, Hoyos, Santalla et al., 2002); professional cyclists have the ability to maintain significantly high percentages of  $\dot{V}O_{2\max}$  for prolonged periods of time (> 60 min) (Lucia et al., 2001; Lucia, Hoyos, Santalla et al., 2002).

Training one's lactate threshold is thought to allow the muscles to work at higher intensities for longer periods before they develop skeletal muscle anaerobiosis (Brooks et al., 2000a; Juel, 1998; Noakes et al., 2001; Walton, Kuchinad, Ivanova, & Garland, 2002). To be competitive, endurance cyclists are required to maintain high exercise intensities for prolonged periods of time (Lucia et al., 1998). Therefore, higher level cyclists are able to exercise at a lower percentage of their  $\dot{V}O_{2\max}$  for a given sub-maximal workload (Gissane et al., 1991; Lucia, Hoyos, Margarita et al., 2002). It is debatable, however, as to whether these adaptations directly reduce the onset of fatigue or occur in response to other adaptations (Noakes, 2000b; Walton et al., 2002).

#### **2.2.4 Summary**

The cardiovascular/anaerobic model of fatigue states that exercise performance is limited by both the ability of the heart to supply sufficient oxygenated blood to the working muscles and the ability of the cardiovascular system to remove accumulated metabolites (Figure 1). A model that is related to the cardiovascular/anaerobic model is the energy supply/energy depletion model.



**Figure 1.** Cardiovascular/anaerobic model of fatigue holds that exercise performance may be limited by the ability of the heart to supply sufficient oxygenated blood to the working muscles. A limited blood supply along with the ability of the muscle to the utilize oxygen (muscle fiber composition) may result in greater anaerobic metabolism. Cycling performance is then inhibited by the build-up of anaerobic metabolites (i.e. hydrogen ions) in the working muscles.



## **2.3 Energy Supply/Energy Depletion Model**

### **2.3.1 Energy supply/metabolic capacity**

The energy supply model proposes that fatigue is a direct consequence of a failure to supply sufficient adenosine triphosphate (ATP) via the various metabolic pathways (phosphocreatine, anaerobic glycolysis, aerobic glycolysis and aerobic lipolysis) (Green, 1997; Noakes, 2000b; Shulman & Rothman, 2001) to the working muscles (Allman & Rice, 2002; Davis & Bailey, 1997; Noakes, 2000b; Shulman & Rothman, 2001). This model is supported by the fact that regular exercise training upregulates the enzymes associated with these energy systems (Hellsten, Apple, & Sjodin, 1996; Kirkenall & Garrett, 1998; Paasuke et al., 1999; Wojtaszewski & Richter, 1998), including creatine kinase (Hellsten et al., 1996), succinate dehydrogenase, and malate dehydrogenase (Burke, Cerny, Costill, & Fink, 1977). For example, Hellsten et al. (1996) showed that seven weeks of sprint cycle training resulted in significant increases in the antioxidant enzymes glutathione peroxidase (GPX) (+36%) and glutathione reductase (GR) (+55%) and metabolic enzyme creatine kinase (CK) (+12.6%). It has been suggested that the decrease in glycolytic and oxidative capacity that naturally occurs as we age may be the cause of increased fatigue onset in aged subjects (Allman & Rice, 2002; Kirkenall & Garrett, 1998). Again however, it is uncertain as to whether or not these adaptations occur as a result of training/detraining, or in response to other adaptations. Indeed, intramuscular ATP stores are highly protected and rarely fall below 40% of resting levels following high-intensity exercise (Green, 1997). This sheds some doubt as to the direct influence that levels of ATP play in determining fatigue. It is unknown whether fatigue might be triggered by certain receptors that would respond to abnormal levels of ATP, but perhaps this could influence afferent nerve fibres or neurotransmitters (Davis, Alderson, & Welsh, 2000).

### **2.3.2 Energy depletion**

The energy depletion model is related to the energy supply model and states that fatigue during prolonged exercise is caused by depletion of muscle and liver glycogen

stores (Brooks et al., 2000b) as well as blood glucose and phosphocreatine (Coyle & Montain, 1992; Lucia et al., 2001; St Clair Gibson, Schabort, & Noakes, 2001). This model is supported by the fact that levels of phosphocreatine are almost completely depleted during short-term high-intensity sprint cycling (Brooks et al., 2000b; Green, 1997; McArdle et al., 2001; St Clair Gibson, Schabort et al., 2001), as are levels of muscle and liver glycogen during prolonged endurance exercise (Brooks et al., 2000b; Dennis et al., 1997). A study performed by Gigli and Bussmann (2002) showed that compared to a control group, glycogen content of rat gastrocnemius muscle decreased by 60% and 86% during 90-min and exhaustive (~3 h) running, respectively. Significant reductions (51%) in ATP concentration were only found in the exhaustive group. It is important to note that exercise must be terminated prior to complete exhaustion of ATP stores, otherwise rigor mortis would occur (Noakes, 2000b).

The metabolic demand of prolonged cycling causes extreme calorie expenditure, requiring significant contributions from carbohydrate, lipid and protein metabolism (Jeukendrup et al., 2000; Mena, Mayner, & Campillo, 1991). The ability to oxidise lipids during endurance exercise is one of the main adaptations that occurs following endurance exercise training (Wojtaszewski & Richter, 1998). This is a beneficial adaptation, as the oxidation of lipid provides nearly 2 ½ times the ATP quantity compared to carbohydrate. The increased rates of fat oxidation at submaximal exercise intensities is clearly of benefit to endurance cyclists as it permits work to be done without depleting carbohydrate stores (Stephens et al., 2001). As the oxidation of carbohydrate permits high rates of ATP production (Dennis et al., 1997; Stephens et al., 2001), while fat and protein do not, our focus in this review will be on the important role that carbohydrate plays in increasing fatigue resistance during prolonged high-intensity exercise.

#### 2.3.2.1 Carbohydrate

Carbohydrate is considered to be the most important fuel source during prolonged exercise and supplies on average  $4.03 \text{ kcal}\cdot\text{g}^{-1}$  (McArdle et al., 2001) at a steady rate of  $340 \text{ }\mu\text{mol}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  ( $86\% \dot{V}\text{O}_{2\text{max}}$ ). For example, the high-intensity exercise performed during time trials ( $>90\% \dot{V}\text{O}_{2\text{max}}$ ) [in the Tour de France]

(Jeukendrup et al., 2000; Lucia et al., 2001), is thought to require significant energy contributions from carbohydrate (Hawley, 2002; Stepto et al., 2001). Indeed, studies have shown that during the later stages of high-intensity cycling protocols ( $>85\% \dot{V}O_{2\max}$ ), carbohydrate oxidation contributes at least 84% of the energy requirements (Stepto et al., 2001). During sub-maximal exercise lasting at least one hour, muscle glycogen stores may decline (Brooks et al., 2000b; Dennis et al., 1997) by 61% (Hawley, Palmer, & Noakes, 1997). It has been found that muscle glycogen may be utilised at a rate of  $4.6 \text{ mmol}\cdot\text{kg dry mass}^{-1}\cdot\text{min}^{-1}$  during a one hour time trial ( $>40.18\text{km}\cdot\text{h}^{-1}$ ) (Hawley et al., 1997) or as high as  $6.5 \text{ mmol}\cdot\text{kg dry mass}^{-1}\cdot\text{min}^{-1}$  during aerobic interval training (5-min at  $86\% \dot{V}O_{2\max}$ , 1-min rest), equating to 90% of the total carbohydrate oxidation (Stepto et al., 2001). The ability of the liver to maintain blood glucose during exercise is therefore dependant upon glycogen stores and upon the activity of stimulating hepatic glycogenolytic and gluconeogenic enzymes (Brooks et al., 2000a; Noakes, 2000b). Due to the limited supply of endogenous carbohydrate, carbohydrate should be the main fuel source consumed during prolonged exercise (Coyle & Montain, 1992). The main purpose of carbohydrate ingestion during prolonged exercise is to maintain blood glucose to permit carbohydrate metabolism late into exercise (Coyle & Montain, 1992; Dennis et al., 1997).

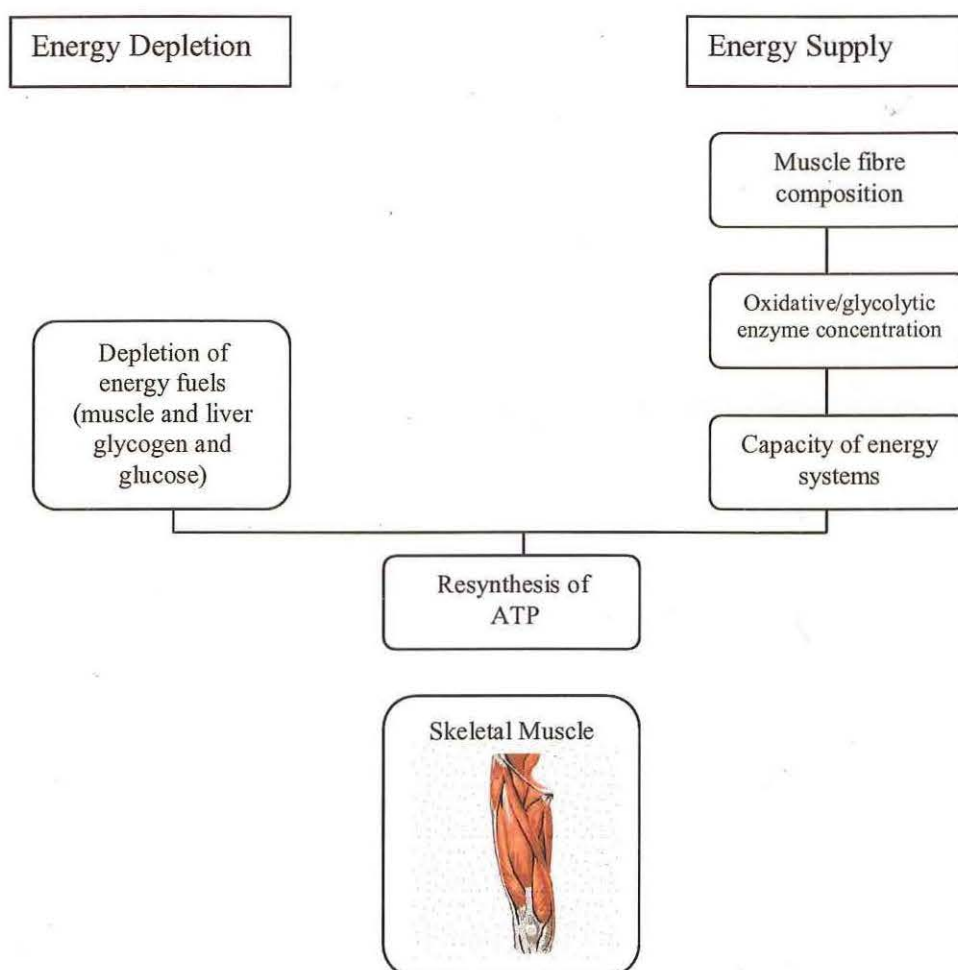
In order for cyclists to maintain power during prolonged cycling it is vital that they consume sufficient carbohydrate. Ingestion of approximately  $30\text{-}60\text{g CHO}\cdot\text{h}^{-1}$  (Coyle & Montain, 1992; Lucia et al., 2001) or  $0.2\text{-}0.6 \text{ g CHO}\cdot\text{kg}\cdot\text{h}^{-1}$  (Applegate, 1989) has been recommended. This can be done in the form of a 5-10% glucose polymer solution (St Clair Gibson, Schabort et al., 2001), at the rate of  $15 \text{ mL}\cdot\text{kgBM}^{-1}\cdot\text{h}^{-1}$  in order to provide glucose at a rate of approximately  $1 \text{ g}\cdot\text{kgBM}^{-1}\cdot\text{h}^{-1}$  (the maximum rate at which muscle can utilise carbohydrate) (St Clair Gibson, Schabort et al., 2001). Furthermore, it has been stated that 45-60 g of carbohydrate ingested at a rate of  $1 \text{ g}\cdot\text{min}^{-1}$  maintains blood glucose levels late in exercise and may delay fatigue for up to 45-min (Coyle & Montain, 1992; Dennis et al., 1997). Interestingly, however, a recent study has shown that the voluntary intake of carbohydrate during strenuous exercise tends to be considerably lower (approximately  $25 \text{ g}\cdot\text{h}^{-1}$ ) (Lucia et al., 2001). If sufficient

carbohydrate is not supplied during prolonged cycling (>2 hr), a greater shift in substrate oxidation from carbohydrate to lipid metabolism occurs as carbohydrate storage becomes depleted (Lepers et al., 2000; Wojtaszewski & Richter, 1998). This reduces the rate of ATP available to working muscle, and is related to fatigue.

It has been found that the ingestion of carbohydrate before and during prolonged exercise can increase cycling time to fatigue and decrease performance time in cycling time trials (Dennis et al., 1997; St Clair Gibson, Schabort et al., 2001). There is also a strong relationship between glycogen levels prior to exercise and time to exhaustion (Brooks et al., 2000b; Coyle & Montain, 1992; Dennis et al., 1997; Kay & Marino, 2000; Shulman & Rothman, 2001). It is for this reason that carbohydrate loading has long been considered to be an ergogenic aid (Burke & Deakin, 2000; Dennis et al., 1997; Hawley et al., 1997). While, the relationship between glycogen and exercise performance is commonly considered to be causally linked at the point of exhaustion (Dennis et al., 1997; Hawley, 2002), muscle glycogen depletion is not considered to be the sole cause of fatigue in prolonged exercise (Dennis et al., 1997; Noakes, 2000b; St Clair Gibson, Schabort et al., 2001). This is because during exhaustive exercise it has been found that skeletal muscle glycogen stores remain high (~60% of resting values) (Fitts, 1994; Kayser, 2003). What is more, a study performed by Hawley et al. (1997) showed that increased muscle glycogen via carbohydrate supplementation had no effect on one-hour cycling time trial performance, muscle glycogen utilisation or lipid oxidation. It has been suggested that perhaps muscle glycogen depletion is a determinant of fatigue in prolonged exercise (>2-3 h) but via another mechanism detached from its role in energy production (Fitts, 1994; Noakes, 2000b). This is supported by Shulman and Rothman's (2001) glycogen shunt model, which suggests that as glycogen concentration is lowered, but still nonzero, there is insufficient muscle glycogen to compensate the millisecond energy demands. Thus, many researchers believe that the oxidative capacity of carbohydrate is not limited by the availability of ATP in the blood (blood glucose) but by the rate at which muscle glucose can be oxidised (energy supply model) (Brooks et al., 2000b; Dennis et al., 1997; Noakes, 2000b).

### 2.3.3 Summary of the energy supply/energy depletion model

The energy supply/energy depletion model involves two mechanisms that may be causal for fatigue during prolonged exercise. The model states that fatigue is related to either an inadequate supply of ATP by the energy systems (energy supply) to working muscle, or that fatigue is the result of an inability to deliver sufficient exogenous/endogenous substrates to the working muscles (energy depletion) (Figure 2).



**Figure 2.** Energy supply/energy depletion model of fatigue states that cycling performance is limited by the availability of adenosine triphosphate (ATP). The inability of the energy systems (phosphocreatine, anaerobic glycolysis, aerobic glycolysis and lipolysis) to resynthesis ATP may inhibit muscle contractions. The depletion of energy stores and enzymes (i.e. phosphocreatine (PC) and creatine kinase (CK)) associated with ATP resynthesis may also reduce cycling performance.

## **2.4 Neuromuscular Fatigue Model**

The majority of past research into the development of fatigue during prolonged exercise has assumed that exercise performance is determined by the ability of the cardiovascular system to deliver and remove substrates to and from the working muscles (Brooks et al., 2000a; Noakes, 2000b). Thus, prolonged endurance exercise performance has traditionally been considered to be limited by either the ability of the cardiovascular system to provide enough blood, nutrients, and/or oxygen to the working muscles, or the ability of the energy systems to supply sufficient ATP to the muscles (Brooks et al., 2000a; Lucia, Hoyos, Santalla et al., 2002; Noakes, 2000b). Although these factors previously discussed are strongly related to fatigue, another theory that has been postulated is that the functions involved in muscle excitation, recruitment and contraction limit performance (Avela, Kyrolainen, & Komi, 2001; Davis et al., 2000; Millet, Lepers et al., 2002; Noakes, 2000b; Stackhouse, Dean, & Lee, 2000). Electromyography (EMG) is commonly used as a global indicator of these factors.

### **2.4.1 Electromyography (EMG) and fatigue**

Electromyography is a measure of both the quality and quantity of electrical muscle activation (Hug, Laplaud, Savin, & GrAlot, 2003; Hunter, St Clair Gibson, Lambert, & Noakes, 2002; Kay et al., 2001; Kay et al., 2000; Lucia et al., 1998). EMG has long been considered an acceptable method of determining functional and dysfunctional recruitment patterns during dynamic movement (Ebenbichler et al., 2002; Hausswirth, Brisswalter, Vallier, Smith, & Lepers, 2000; Hug, Laplaud et al., 2003; Hunter, St Clair Gibson, Lambert et al., 2002; Jammes et al., 2000; Kay et al., 2000; Lucia et al., 1998). During fatiguing exercise, there tends to be a gradual decrease in EMG activity by the working muscles (Ebenbichler et al., 2002; Enoka, Rankin, Joyner, & Stuart, 1988; Farina, Fattorini, Felici, & Filligoi, 2002; Jammes et al., 2000; Lucia et al., 1998; Sacco, Newberry, McFadden, Brown, & McComas, 1997). A reduction in the firing frequency of motoneurons, along with an increase in the time taken for muscle fibre relaxation has been recognised as the fundamental attributing factor to this decrease in performance (Pinniger et al., 2000; Sacco et al., 1997). Thus, it has been well

established that analysis of changes in the integrated electromyographic signal (iEMG), or root mean square (RMS), and the compound action potential (M-wave) is an effective measure of neuromuscular fatigue (Hauswirth et al., 2000; Hautier et al., 2000; Hug, Decherchi, Marqueste, & Jammes, 2004; Jammes et al., 2000; Lepers, Millet, & Maffiuletti, 2001). Whereby, iEMG refers to an EMG signal which has been filtered and smoothed (Loeb & Gans, 1986), the RMS is a quantification of the global EMG signal (Cram & Kasman, 1998; Hug, Laplaud et al., 2003) and action potential refers to the electrical impulse sent from the brain (Loeb & Gans, 1986). Central activation failure has been associated with a reduction in the RMS irrespectively of maintained M-wave amplitude (Lepers, Millet, & Maffiuletti, 2001).

It is debatable as to where on the pathway from the central nervous system to the peripheral contractile mechanism the neuromuscular fatigue response is controlled (Lepers et al., 2000; Lepers et al., 2002; Lepers, Millet, & Maffiuletti, 2001). In fact, numerous studies into neuromuscular function during prolonged cycling have suggested that a reduction in leg muscle capacity occurs as a result of alterations to both central and peripheral mechanisms (Table 1) (Gabriel et al., 2001; Hautier et al., 2000; Kay et al., 2001; Lepers et al., 2000; Lepers et al., 2002; Lepers, Millet, & Maffiuletti, 2001; Millet et al., 2003; Nybo & Nielsen, 2001a). This hypothesis holds the basis for the neuromuscular fatigue model (Bilodeau et al., 2001; Davis & Bailey, 1997; St Clair Gibson, Schabort et al., 2001).

#### **2.4.2 Neuromuscular fatigue**

Neuromuscular fatigue refers to a reduction in the force (Avela et al., 2001; Kay et al., 2000; Lepers et al., 2000; Lepers et al., 2002; Lunde, Verburg, Vollestad, & Sejersted, 1998; Millet, Lepers et al., 2002; Millet et al., 2003; Paasuke et al., 1999; Rodacki et al., 2001; St Clair Gibson, Lambert et al., 2001) or power (Allman & Rice, 2002; Hautier et al., 2000) production of a muscle despite a greater perception of effort (Gollhofer, Komi, Miyashita, & Aura, 1987; Hampson et al., 2001; Kay et al., 2000; St Clair Gibson, Lambert et al., 2001). While an abundance of research is available on the effects of exercise upon neuromuscular function (Gollhofer, Komi, Miyashita et al., 1987; Lepers et al., 2000; Lepers et al., 2002; Nybo & Nielsen, 2001a; Pinniger et al.,

2000; Rodacki et al., 2001; St Clair Gibson, Lambert et al., 2001; Takaishi, Yasuda, Ono, & Moritani, 1996) few studies have examined the relationship between neuromuscular function and prolonged cycling exercise (Lepers et al., 2000; Lepers et al., 2002; St Clair Gibson, Schabert et al., 2001). Focal to the neuromuscular fatigue model are two reviewed philosophies explaining the resulting decrease in neural drive that occurs during exercise. These ideas are primarily based upon where, in the chain of command, from the motor centres in the brain to the actin-myosin crossbridging, failure or impairment occurs (Avela et al., 2001; Davis, 1995; Lepers, Millet, & Maffiuletti, 2001; Paasuke et al., 1999). These concepts include, the central activation failure theory (Allman & Rice, 2002; Lepers, Millet, & Maffiuletti, 2001; Millet et al., 2003) and the neuromuscular propagation failure theory (Allman & Rice, 2002).

#### **2.4.2.1 Central activation failure theory**

Traditionally, studies into neuromuscular fatigue during exercise have focused upon alterations in the neuromuscular junction or in the muscles themselves, with little attention paid to the central nervous system (Davis et al., 2000; Davis & Bailey, 1997; Schillings, Hoefsloot, Stegeman, & Zwarts, 2003). The cause of central fatigue, defined as a progressive diminution of the muscle activation by the central nervous system, is still inconclusive (Allman & Rice, 2002; Avela et al., 2001; Davis et al., 2000; Davis & Bailey, 1997; Green, 1997; Kay et al., 2001). Research has found that after prolonged ultramarathon exercise, maximal voluntary contractions (MVC) of the quadriceps muscles decrease by  $32\% \pm 18\%$  (Millet, Lepers et al., 2002). A 10% reduction in maximal voluntary contraction of the leg extensors was found after a prolonged cycling race (140km at  $31\text{km}\cdot\text{hr}^{-1}$ ) (Millet, Lepers et al., 2002). This change in MVC is thought by some to be due to a reduction in neural input (Kay & Marino, 2000; Kay et al., 2000; Millet, Lepers et al., 2002; Sacco et al., 1997) resulting from the control of a “central governor” located in the brain (Noakes et al., 2001; St Clair Gibson, Lambert et al., 2001) associated with a build up of intracortical inhibition in response to the development of pain (Paasuke et al., 1999).

As fatigue develops during prolonged exercise, there is an increase in intracortical inhibition (Millet et al., 2003; Paasuke et al., 1999). It has been suggested



that increases in the neurotransmitter serotonin or 5-hydroxytryptamine and perhaps dopamine and acetylcholine concentrations in the brain reduce the rate of central neural drive, which determine the excitement and recruitment of skeletal muscle (Davis, 1995; Davis et al., 2000; Davis & Bailey, 1997; Kay & Marino, 2000). It is thought that these transmitters have an important effect upon arousal, lethargy, sleepiness, and mood, which in turn may effect perception of effort during exercise (Davis & Bailey, 1997). Indeed, it has been shown that an artificial increase of dopamine (Davis & Bailey, 1997) and serotonin (Davis et al., 2000; Davis & Bailey, 1997) in the brain reduces exercise performance. However, Bailey et al. (1993) showed that during prolonged exercise in rats, fatigue was associated with increases in serotonin and decreases in dopamine concentrations in the brain. It has since been hypothesized that a low serotonin/dopamine ratio may improve muscle activation (via increasing arousal, motivation and optimal neuromuscular coordination) and therefore improve prolonged exercise performance (Davis et al., 2000). What is more, it has been suggested that nutritional status (especially that of carbohydrates) may affect neurotransmitters (i.e., attenuated increases in 5-hydroxytryptamine) (Davis et al., 2000), and consequently central fatigue may be related to the energy supply/energy depletion model. More research is necessary, however, in order to distinguish between the specific effects of carbohydrate ingestion on central activation as apposed to its well known advantageous peripheral effects (Davis et al., 2000).

Few studies that have assessed EMG during cycling have looked specifically at central fatigue (Lepers et al., 2000; Lepers, Millet, & Maffiuletti, 2001). Of these studies, the involvement of the central nervous system in fatigue is often determined via twitch interpolation (where stimuli (single, double or tetanic) are sent to the motor axons thus removing the central nervous system from the contraction) is compared to voluntary contractions (Allman & Rice, 2002; Avela et al., 2001; Lepers et al., 2000; Lepers, Millet, & Maffiuletti, 2001; Stackhouse et al., 2000). If no difference is found then it is assumed that no central fatigue has occurred (Allman & Rice, 2002; Davis et al., 2000; Davis & Bailey, 1997; Paasuke et al., 1999). A major flaw in this method of determining central fatigue is that many other factors including psychological (de Ruiter, Jongen, van

der Woude, & de Haan, 2001; Hampson et al., 2001; Kay & Marino, 2000; Kay et al., 2001; O'Brien & O'Connor, 2000; O'Connor & Cook, 2001; Tikuisis, McLellan, & Selkirk, 2002), environmental (Kay & Marino, 2000; Nybo & Nielsen, 2001a; Tikuisis et al., 2002) and hydration level (Lepers et al., 2000; Paasuke et al., 1999; Schillings et al., 2003) have all been shown to effect neural drive and thus exercise performance. An extension upon previous methods of determining central activation failure was developed by Schillings et al. (2003) who compared the superimposed force (via 5-pulse-train twitch interpolation every 15 seconds) with estimated force decline due to peripheral factors (muscle fibre conduction velocity). Under these circumstances, a decline in muscle fibre conduction velocity reflects transformations within the muscle (i.e., the accumulation of metabolic byproducts) and thus relates to peripheral fatigue (Schillings et al., 2003). Using twitch interpolation (12-pulse electrical train), Stackhouse et al. (2001) found a lower central activation ratio (ratio of voluntary force production to total force production using superimposition) in an isometric exercise-induced fatigued state (0.90, 0.74) compared to a non-fatigued state (0.98, 0.94), in young and elderly subjects respectively. The lower central activation ratio in elderly subjects under both conditions suggests a greater central activation failure, which may be related to age-related weakness (Stackhouse, Stevens et al., 2001). In summary, as neural drive may significantly affect exercise performance, determination of central activation failure allows the separation of cerebral alterations (i.e., neurotransmitters and/or cognitive alterations) and peripheral changes that may lead to fatigue.

#### **2.4.2.2 Neuromuscular propagation failure theory**

An alternate notion explaining a decrease in force production is that peripheral mechanisms could be involved (Allman & Rice, 2002; Enoka et al., 1988; Green, 1997; Lucia et al., 1998; Nielsen & Clausen, 2000). This theory holds that the ability of the muscle to produce force is limited by the response of the muscle to an electrical stimulus (Fowles, Green, Tupling, O'Brien, & Roy, 2002; Lepers et al., 2002; Sacco et al., 1997; Walton et al., 2002). Thus, alterations in the muscle action potential (M-wave) may reflect a decrease in membrane excitability (Brooks et al., 2000b; Fowles et al., 2002; Jammes et al., 2000; Millet, Lepers et al., 2002). In fact, an inhibition is thought to occur

at the sarcolemma (Fowles et al., 2002; Gollhofer, Komi, Miyashita et al., 1987; Green, 1997; Millet, Lepers et al., 2002; Sacco et al., 1997) or  $\alpha$ -motoneuron (Avela et al., 2001; Hunter et al., 2004; Sacco et al., 1997; Walton et al., 2002), whereby a peripheral reflex response originates in the functioning muscles causing a reduction in neural activation (Avela et al., 2001; Fowles et al., 2002; Kay & Marino, 2000; Sacco et al., 1997). Interestingly, it has been found that during submaximal isometric contractions (15% of MVC) whilst performing a positioning task compared to a force task there is greater motor unit activity, causing greater variation in motor output, mean arterial pressure, heart rate and ratings of perceived exertion (Hunter, Ryan, Ortega, & Enoka, 2002). The position task requires a subject to maintain a given elbow angle whilst supporting a submaximal load and the force task required the subject to apply a submaximal force (equal to that of the position task) against an immovable object (Hunter et al., 2004; Hunter, Ryan et al., 2002). It has since been suggested that the reduced time to failure in the position task (50% of the force trial), despite similar rates of increases in EMG, resulted from greater excitatory and inhibitory input to the motor neuron pool (Hunter et al., 2004; Hunter, Ryan et al., 2002). This then highlights the task specific nature of fatigue and that differing mechanisms are responsible for “task failure” under varying exercise conditions (Hunter et al., 2004).

During prolonged exercise, reductions in ionic ( $\text{Na}^+$  and  $\text{K}^+$ ) transmembrane gradients may occur, resulting in a decreased muscle action potential (M-wave) (Fowles et al., 2002; Green, 1997; Harnada, Sale, MacDougall, & Tarnopolsky, 2003; Nielsen & Clausen, 2000). In short, a reduction in conduction velocity of action potentials, alterations to M-wave amplitude and/or a decrease in pH have all been found to be related to reduced EMG activity during prolonged cycling exercise (Allman & Rice, 2002; Fowles et al., 2002; Green, 1997; Jammes et al., 2000; Kay et al., 2000; St Clair Gibson, Lambert et al., 2001). Furthermore, increases in intracellular lactate and in extracellular  $\text{K}^+$  results in a decrease in membrane excitability and a reduction in central activation (Green, 1997; Oba, Ishikawa, Takaishi, Aoki, & Yamaguchi, 2000). The increased intracellular  $\text{Na}^+$  (Nielsen & Clausen, 2000) and reduced intracellular  $\text{K}^+$  has been attributed to insufficient activation of the  $\text{Na}^+/\text{K}^+$  muscle pumps (Fowles et al., 2002; Green, 1997; McKenna, Harmer, Fraser, & Li, 1996; Nielsen & Clausen, 2000;

Walton et al., 2002). Evidence suggesting that neuromuscular propagation failure may be a limiting factor in prolonged cycling performance comes from the fact that submaximal endurance training (cycling 5-6 times per week at 68%  $\dot{V}O_{2peak}$ ) resulted in a 35% increase in  $Na^+/K^+$  pump concentration of the vastus lateralis (Green et al., 1999). As group III and IV muscle receptors have been found to react to fatigue-related changes (i.e., anoxia, increased  $H^+$  or  $K^+$  and/or the production of neuropeptides) within the muscle, then these receptors are thought to be influential upon the peripheral reflex fatigue response (Lepers et al., 2000; Lepers et al., 2002; Ray & Gracey, 1997; St Clair Gibson, Lambert et al., 2001; Walton et al., 2002). However, Walton et al. (2002) found that during submaximal isometric plantarflexion contractions (30% of MVIC), reductions in reflex amplitude were not exclusively the result of group III and IV's chemical sensitivity to fatigue-related muscular waste products. This is supported by Ray and Gracey (Ray & Gracey, 1997), who found that under thermally elevated conditions, muscular sympathetic activity can increase due to an increase in the firing rate of group III and IV afferents. A decrement in M-wave peak to peak amplitude has been found in a study of well-trained male cyclists that performed 2-hours of cycling at 65% maximal aerobic power (Lepers et al., 2000). However, no significant changes in M-wave amplitude were found during fatiguing sub-maximal cycling protocols lasting only 30-minutes (Bigard et al., 2001; Lepers et al., 2000). This suggests that there is a relationship between the specific task performed and the fatigue response during exercise.

In summary, neuromuscular propagation failure theory states that neuromuscular fatigue occurs due to a reduced response of the muscle to an electrical stimulus at the level of the sarcolemma or  $\alpha$ -motoneuron.

#### **2.4.2.3 Muscle power model/peripheral failure theory**

Fatigue is also thought to occur directly at the muscle level, whereby alterations in the coupling mechanism between action potential and contractile proteins (Brooks et al., 2000b; Enoka et al., 1988; Hill et al., 2001; Oba et al., 2000) or calcium release, calcium regulation at actin-myosin contractile level, cross-bridge cycling (Brooks et al., 2000b; Hill et al., 2001; Kay et al., 2001; Noakes, 2000b; Paasuke et al., 1999;

Schillings et al., 2003) or depletion of energy stores occurs (Kay & Marino, 2000). Much of the research into peripheral failure has looked at reductions in maximal voluntary contractions (Balmer et al., 2000; Bigard et al., 2001; de Ruiter et al., 2001; Ebenbichler et al., 2002; Gabriel et al., 2001; Gollhofer, Komi, Fujitsuka, & Miyashita, 1987; Gollhofer, Komi, Miyashita et al., 1987; Hausswirth et al., 2000; Hunter, St Clair Gibson, Lambert et al., 2002; Lepers et al., 2000; St Clair Gibson, Lambert et al., 2001). However, it has been suggested that prolonged exercise may have a greater initial impact upon muscular endurance rather than muscular strength (Kay et al., 2000; Lucia et al., 1998). In fact, it has been found that during both maximal and sub-maximal exercise, central activation (EMG firing rate) may be increased in order to achieve the same torque or power (Allman & Rice, 2002; Hermann & Barnes, 2001; Lucia et al., 1998). This disproportional relationship between motor unit recruitment and force production (known as the fatigue or EMG threshold) is reflective of a compensation for the diminishing peripheral force production caused by fatiguing motor units (Lucia et al., 1998; Pringle & Jones, 2002). Interestingly, through the use of fine-wire and surface electrodes, Westgaard and de Luca (1999) found that during a number of contractions (static, manipulation and operational task) lasting 10-min, low-threshold motor units had periods of inactivity after the first few minutes of the contraction. During these periods, higher-threshold motor units were recruited and surface EMG (action potential) remained constant. This “substitution” process may be reflective of a protective mechanism in order to defend the individual motor units against complete exhaustion (Westgaard & de Luca, 1999), thus supporting the existence of muscle power/peripheral failure.

Low-frequency fatigue has been related to a reduction in calcium ( $\text{Ca}^{2+}$ ) release to a given stimulus (Bilodeau et al., 2001; Green, 1997; Hill et al., 2001; Lepers et al., 2000; Lepers et al., 2002; Lepers, Millet, & Maffiuletti, 2001; Schillings et al., 2003).  $\text{Ca}^{2+}$  release from the sarcoplasm reticulum is vital for muscle contraction (McKenna et al., 1996). It is thought that a reduction in  $\text{Ca}^{2+}$  release from the sarcoplasm reticulum, caused by increases in lactate anion and/or inorganic phosphate concentration, may negatively influence the excitation-contraction coupling process (Brooks et al., 2000b; Fowles et al., 2002; Green, 1997; Hamlin & Quigley, 2001b; McKenna et al., 1996;

Stackhouse, Reisman et al., 2001). During fatiguing exercise there may also be a reduction in  $\text{Ca}^{2+}$  return from contractile proteins to the sarcoplasmic reticulum, which may be responsible for an increase in the muscle relaxation time (Hill et al., 2001; McKenna et al., 1996). The response of contractile elements to free calcium may also be delayed (Green, 1997; Hamlin & Quigley, 2001a). During excitation-contraction coupling, the calcium released by the sarcoplasmic reticulum may also be taken up by the mitochondria, which may interfere with mitochondrial function (Brooks et al., 2000b; Davis & Bailey, 1997). An increase in mitochondrial  $\text{Ca}^{2+}$  causes greater oxygen consumption and reduces the phosphorylation ability of ADP to ATP (Brooks et al., 2000b). It is also important to note that researchers often fail to address alterations in muscle power mechanics, and therefore view peripheral fatigue as the combination of neuromuscular propagation failure and changes at the level of the muscle (Enoka et al., 1988; Hunter, St Clair Gibson, Lambert et al., 2002; Kay et al., 2001; Kay et al., 2000; Wilmore & Costill, 1999).

As previously stated, the use of electrical stimulation (trains of five stimuli every 15 s) was used by Schillings et al. (2003) who found that during a 2-min maximal contraction of the biceps brachii, the decrease in voluntary force (38%) in the first minute was largely the result of peripheral factors (89%), after which further decrements in force were a result of mainly central fatigue. This pattern makes sense as demands are initially placed upon the muscle, with the latter sequence resulting from a number of alterations occurring in the central nervous system. This suggests that there may be multiple fatigue sites depending on the physiological requirements of the task. It has also been hypothesised that this relationship between neural and peripheral fatigue is a safety mechanism (Gabriel et al., 2001; Pinniger et al., 2000; St Clair Gibson, Lambert et al., 2001), whereby motor unit firing rate is reduced by the central nervous system in order to protect  $\text{Na}^+$ , ATP concentration (Kay et al., 2001; St Clair Gibson, Schabort et al., 2001), and to avoid excessive damage of the muscle fibres (Noakes, 2000b; St Clair Gibson, Lambert et al., 2001). Thus, central fatigue could be the response to afferent input from peripheral organs in order to prevent injury or death by causing a reduction or termination of activity (Allman & Rice, 2002; Kay & Marino, 2000; Kay et al., 2001; Lepers et al., 2000; Paasuke et al., 1999; St Clair Gibson, Lambert et al., 2001; St Clair

Gibson, Schabert et al., 2001). To date, however, it is inconclusive as to whether or not the same patterns of fatigue occur during prolonged sub-maximal cycling.

#### **2.4.3 Summary of the neuromuscular fatigue model**

In relation to the muscle recruitment fatigue model, there are three viewpoints that explain the resulting decrease in muscle activation and contraction during prolonged cycling. These models have been developed to explain where, along the neuromuscular pathway, inhibition occurs (Figure 3). While the central activation failure theory involves a reduction in the neural drive, the neuromuscular propagation failure theory sees fatigue as a result of reduced responsiveness of the muscle to an electrical stimulus. Finally, the muscle power/peripheral failure theory states that fatigue occurs within the muscle and involves excitation-contraction coupling mechanisms.

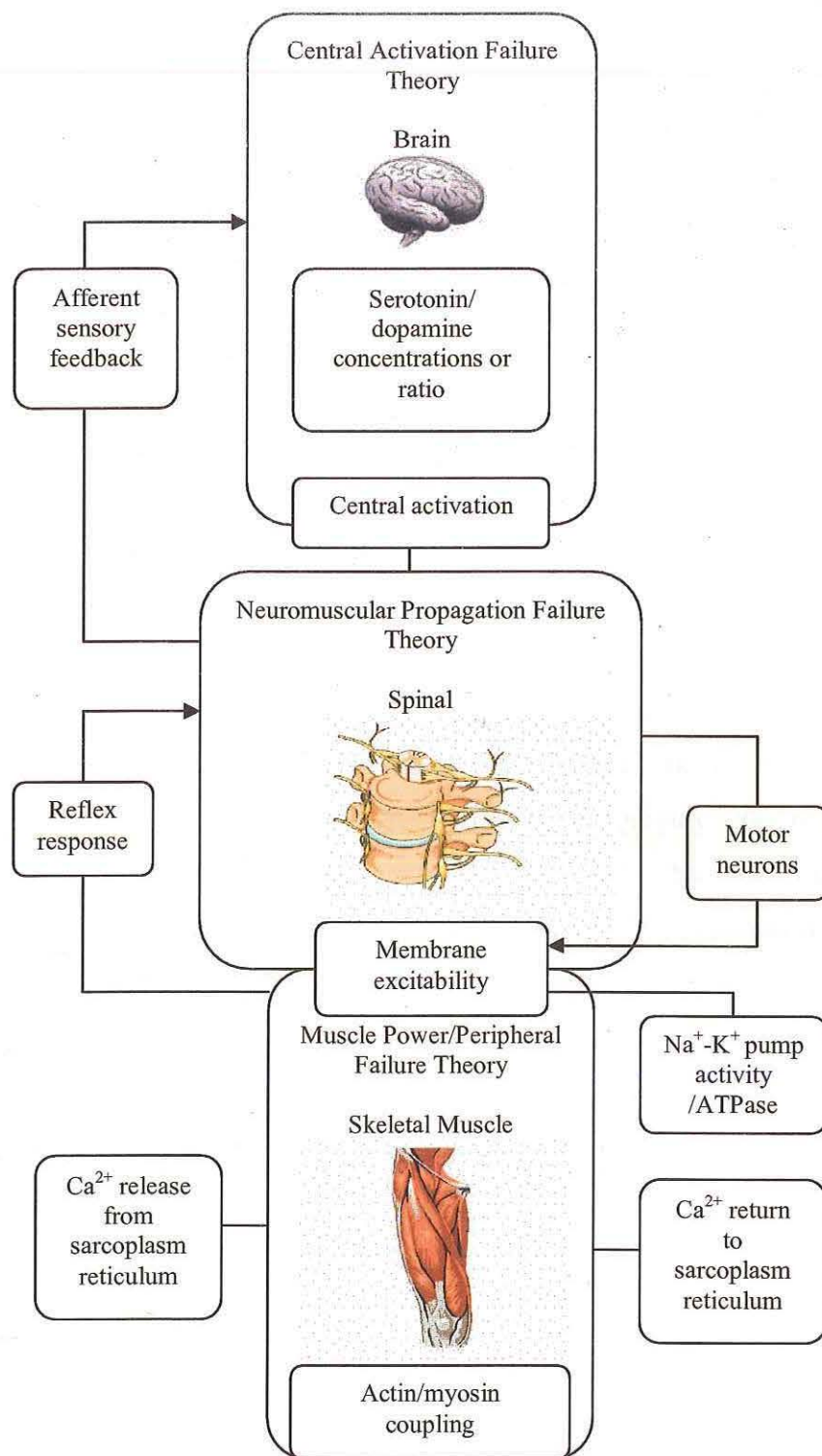
**Table 1.**

Alterations in central activation RMS and M-wave properties in cross-sectional cycling studies

Reference	Activity	Reduction in Central activation	Muscle	Increase in M-wave duration	Reduction in M-wave amplitude	Reduction in RMS/ RMS <sub>M</sub>
(Hautier et al., 2000)	2-min cycling	-----	VL	-----	13.89%	-----
(Lepers et al., 2000)	30-min cycling	16.10%	VL	insignificant	insignificant	30 +/-5%
			VM	insignificant	insignificant	30 +/-6%
(Lepers, Millet, & Maffiuletti, 2001)	2-h cycling	24.20%	VL	19.60%	14.10%	-----
			VM	10.30%	18.90%	-----
(Lepers et al., 2002)	5-h cycling	8.50%	VL	5.90%	7.80%	16%
(Millet et al., 2003)	140-km cycling	insignificant	RF	-----	-----	
(Nybo & Nielsen, 2001a)	Cycle to exhaustion at 40°C (~60% $\dot{V}O_{2\max}$ )	54%	RF	-----	-----	-----

Summary of cross-sectional studies, that have measured the fatigability of the quadriceps (VL, vastus lateralis; VM, vastus medialis; RF, rectus femoris), during prolonged sub-maximal cycling. Increases in M-wave duration, showing an increased duration of action potential, along with decrease in M-wave amplitude, suggests impairment of neuromuscular propagation and peripheral fatigue (Lepers et al., 2000; Lepers et al., 2002; Lepers, Millet, Maffiuletti et al., 2001). A reduction in RMS/RMS<sub>M</sub>, [root mean square/root mean square (M-wave)] and central activation suggests an impairment of central activation (Lepers et al., 2000; Lepers et al., 2002; Lepers, Millet, Maffiuletti et al., 2001).





**Figure 3.** The neuromuscular fatigue model states that exercise performance may be limited due to a reduced central activation, caused by alterations in neurotransmitter concentrations or in response to afferent sensory feedback. Alternatively, reduced cycling performance may be caused by reduced membrane excitability due to alterations in ionic pump activation and/or the motor neuron pool. Finally, inhibition may occur in the muscle as calcium status may affect actin/myosin function.

## 2.5 Muscle Trauma Model

The stress of exercise-induced muscle damage can have numerous detrimental effects on muscle function, ranging from disruption of the sarcolemma or sarcomere, to complete tear of myofibrils (del Aguila, Claffey, & Kirwan, 1999; Gollhofer, Komi, Miyashita et al., 1987; Komi, 2000; Nosaka, Lavender, Newton, & Sacco, 2003), thus causing alterations in body homeostasis (Hamlin & Quigley, 2001b; Nicol, Kuitunen, H., Avela, & Komi, 2003; Nosaka et al., 2003). Muscle damage can be classified into three differing categories based upon specific clinical changes. Type I injury refers to exercise-induced muscle damage that is associated with muscle swelling, stiffness and delayed onset of muscle soreness (DOMS), occurring 24 to 48 hours after exercise (Hamlin & Quigley, 2001b). Type II injury includes the specific tearing of muscle fibres (Hamlin & Quigley, 2001b). Type III muscle damage refers to muscle soreness and/or cramps that occur during or immediately after exercise (del Aguila et al., 1999; Green, 1997; Hamlin & Quigley, 2001a, 2001b; Nicol et al., 2003; Nosaka et al., 2003).

Much of the research into tissue damage during exercise has looked at the physiological responses to eccentric exercise (Green, 1997; Hamlin & Quigley, 2001a, 2001b; Hermann & Barnes, 2001; Proske & Morgan, 2001). The fact that professional cyclists are required to push high gears (i.e., 53 x 12-11) for prolonged lengths of time, some extent of muscle damage is to be expected (Lucia et al., 2001). Gibala et al. (1995) showed that after a single bout of resistance exercise (8 sets, 8 reps, 80% of 1RM) untrained males exhibited greater myofibrillar disruption of the elbow flexors resulting from eccentric (>80%) compared to concentric (>30%) exercise. However, in a later study using the same protocol on trained individuals the damage following eccentric exercise was found to be significantly lower (eccentric 45%; concentric 27%) (Gibala et al., 2000). In relation to damaged muscle, it is thought that reductions in force production may be the result of alterations to the excitation-contraction coupling system, sarcomeres (Fowles et al., 2002; Green, 1997; Hamlin & Quigley, 2001b; Proske & Morgan, 2001) and intracellular calcium homeostasis (Brooks et al., 2000b; Green, 1997; Proske & Morgan, 2001). The use of oxygen increases during prolonged exercise causing an increase in the release of free radicals (Hellsten et al., 1996; Noakes et al., 2001). Reports suggest that this

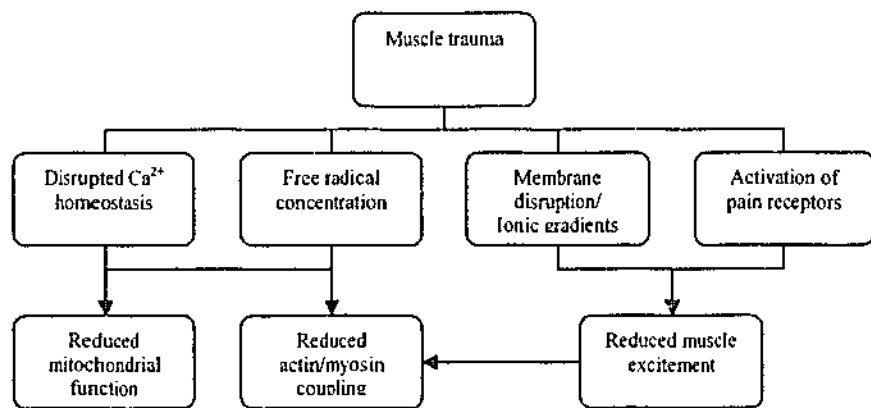
accumulation of free radicals is damaging to mitochondria, resulting in a reduced ability to utilise oxygen, and subsequently a reduced exercise performance (Fowles et al., 2002; Green, 1997). Evidence also shows that damage to free radicals may also reduce the activity of  $\text{Na}^+\text{-K}^+\text{-ATPase}$ , a membrane protein that maintains homeostatic ionic gradients at the sarcolemma (Ciubotariu, Arendt-Nielsen, & Graven-Nielsen, 2004; del Aguila et al., 1999; Hamlin & Quigley, 2001b; Mena, Mayner, & Campillo, 1996; Nicol et al., 2003). What is more, it has also been suggested that damage to the muscle cells may cause a significant increase in plasma aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH) during prolonged cycling (Ciubotariu et al., 2004). The effect that these particular enzymes have on power output and fatigue are, to date, unclear. However, structural and chemical imbalances caused by exercise-induced muscle damage are thought to have an influence upon the neuromuscular/afferent sensory pathways (EMG/force ratios) (Hampson et al., 2001). Through EMG analysis, it has been found that increased muscle pain, caused by the injection of hypertonic saline, resulted in a reduced activation of the muscle agonist, and ultimately increased the activity of the muscle antagonist during sustained submaximal (30% and 80%) isometric contractions (Ciubotariu et al., 2004). This lead to a reduction in endurance time of the pain-induced trials (Ciubotariu et al., 2004). This muscular fatigue is associated with a decreased RMS of pain-induced muscle (lowered EMG/force ratio) and reduced EMG amplitude of non-pain synergistic muscles (Ciubotariu et al., 2004). This suggests that induced pain reduces muscular activation, thus causing fatigue of compensatory synergistic muscles (Ciubotariu et al., 2004). The precise mechanisms responsible for a decrease in performance of pain-induced muscle are unknown, but may be due to the relationship between pain receptors and the CNS (central governor) or an increase in type II fibre recruitment (Hampson et al., 2001; Nicol et al., 2003; Nosaka et al., 2003).

It has been found that after exercise-induced soft tissue damage, there is an increase in insulin secretion and a temporary, entire body, systemic insulin resistance (Brooks et al., 2000b) accompanied with reduced creatine kinase activity (Green, 1997; Noakes et al., 2001). Hence, as insulin affects the rate at which glucose is utilised and creatine kinase affects the rate at which ATP is produced, it is thought that these changes will have significant influence upon performance and fatigue. To

date, the cellular mechanisms responsible for insulin resistance in skeletal muscle are unclear, but may be possibly due to alterations in the insulin signalling pathways (Green, 1997; Kay et al., 2001; Kayser, 2003; Lucia, Hoyos, Santalla, Earnest et al., 2003; St Clair Gibson, Schabort et al., 2001).

### 2.5.1 Summary of the muscle trauma model

In summary, the muscle trauma model states that muscle damage resulting from prolonged cycling may cause a reduction in the power producing capacity of the active muscles. This is because prolonged cycling may be causal for significant disruption to the muscle, causing alterations to intramuscular chemical homeostasis and activation of pain receptors, which in turn may result in reduced neuromuscular activation and/or reduced force production of the muscle (Figure 4).



**Figure 4.** The muscle trauma model states that exercise induced muscle damage causes alterations that may limit prolonged cycling performance. Such alterations include disturbances to ionic pumps leading to disrupted chemical homeostasis. This may, in turn, lead to activation of pain receptors, and ultimately a reduction in actin/myosin coupling.

## 2.6 Biomechanical Model

The biomechanical model of fatigue states that fatigue is governed by the efficiency of movement patterns during exercise (Hauswirth, Bigard, & Guezennec, 1997), and that improved efficiency leads to 1) a reduction in the oxygen

consumption required for a given workload (Gissane et al., 1991; Hahn & Gore, 2001; Passfield & Doust, 2000; Takaishi, Yamamoto, Ono, Ito, & Moritani, 1998; Takaishi et al., 1996), 2) a reduced depletion of energy stores (Hahn & Gore, 2001), 3) a delayed accumulation of metabolites, and 4) an attenuation of the increase of core body temperature (Candau et al., 1999).

### **2.6.1 Efficiency of motion (cycling economy)**

It has been suggested that reductions in performance during prolonged sub-maximal cycling are consistent with fatigue-related decrements in gross efficiency (Hauswirth et al., 1997). A decreased efficiency during repeated submaximal exercise is observed with significant increases in  $\dot{V}O_2$  (Candau et al., 1999; Passfield & Doust, 2000), which is accompanied by a reduction in maximal voluntary force production and power producing capacity of a muscle (Lucia, Hoyos, Margarita et al., 2002). Thus, a comparatively greater economy/efficiency of motion, as reported in professional world-class cyclists (Lucia, Hoyos, Margarita et al., 2002; Lucia, Hoyos, Santalla et al., 2002), may decrease the percentage of  $\dot{V}O_{2\max}$  and anaerobic threshold needed to sustain a given power output (Lepers, Millet, & Maffiuletti, 2001; Lucia et al., 2000; MacIntosh, Neptune, & Horton, 2000; Millet, Tronche, Fuster, & Candau, 2002; Millet, Lepers et al., 2002; Oba et al., 2000), allowing a greater performance during prolonged cycling. It has been reported that cycling economy and gross mechanical efficiency of 11 elite male cyclists averaged  $85.2 \text{ W} \cdot \text{LO}_2^{-1} \cdot \text{min}^{-1}$  and 24.5% (Lucia, Hoyos, Margarita et al., 2002). However, such high values of gross efficiency have sparked much debate (Atkinson, Davison, Passfield, & Nevill, 2003; Jeukendrup, Martin, & Gore, 2003; Lucia, Hoyos, Santalla, & Chicharro, 2003; Lucia, Hoyos, Santalla, Rabadan, & Chicharro, 2003) as gross efficiency during cycling usually ranges from 18-22% (Jeukendrup et al., 2003).

The factors influencing gross efficiency and thus fatigue during exercise are strongly dependant upon the specific exercise task performed (i.e., voluntary vs. electrical stimulation, isometric vs. concentric vs. eccentric, sustained vs. intermittent, maximal vs. sub-maximal force generation and duration) (de Ruiter et al., 2001; Farina et al., 2002; Garside & Doran, 2000; Lepers, Millet, & Maffiuletti, 2001; MacIntosh et al., 2000; Millet, Tronche et al., 2002; Millet, Lepers et al., 2002;

Oba et al., 2000; Paasuke et al., 1999), and muscle properties (Gabriel et al., 2001; Green, 1997; Harnada et al., 2003; Lepers, Millet, & Maffiuletti, 2001; Lucia, Hoyos, Margarita et al., 2002; Paasuke et al., 1999; Weston, Karamizrak, Smith, Noakes, & Myburgh, 1999). In fact, higher percentages of slow twitch muscle fibres, as found in endurance-trained athletes, may result in greater fatigue resistance than their power-trained counterparts (Cherry, Lakomy, Nevill, & Maddox, 1997; Harnada et al., 2003; Lepers, Millet, & Maffiuletti, 2001; Lucia, Hoyos, Margarita et al., 2002; MacIntosh et al., 2000; Takaishi et al., 1998; Takaishi et al., 1996). Researchers have also found that the pedal rate (cadence) strongly affects the rate at which fatigue occurs (Cherry et al., 1997; Lepers, Millet, & Maffiuletti, 2001; MacIntosh et al., 2000; Takaishi et al., 1998; Takaishi et al., 1996). Takaishi et al. (1996) showed during cycling (85%  $\dot{V}O_{2max}$  for 15-min) that trained cyclists tend to select an optimal cadence (80 to 90 rev·min<sup>-1</sup>) which is associated with reduced neuromuscular fatigue. As non-cyclists show a reduced neuromuscular fatigue at 70 rev·min<sup>-1</sup>, it was suggested that the optimal pedalling rate would gradually increase in relation to training status (Takaishi et al., 1998; Takaishi et al., 1996) due to enhanced pedalling biomechanics (Takaishi et al., 1998). An increased cadence is associated with an increased blood flow due to lower blood flow occlusion (Gotshall et al., 1996; Takaishi et al., 1998) and greater cardiac output (Gotshall et al., 1996). Gotshall et al. (1996) found that a cadence of 110 rev·min<sup>-1</sup>, as opposed to 70 or 90 rev·min<sup>-1</sup> (200W) in trained cyclists, resulted in a drop in the arterial-venous O<sub>2</sub> difference, suggesting that the cardiac output was in excess of  $\dot{V}O_2$ . It has been suggested that this increase in blood flow may assist in a greater removal of lactic acid (cardiovascular/anaerobic model) and thus be a contributing factor responsible for the higher cadences chosen by elite cyclists (Gotshall et al., 1996). Force output will also be reduced at higher cadences, resulting in a greater activation of slow twitch muscle fibres (Cherry et al., 1997; Harnada et al., 2003; Lepers, Millet, & Maffiuletti, 2001; Takaishi et al., 1998; Takaishi et al., 1996), which may explain the greater oxygen consumption (reduced cycling economy) found at higher cadences (Cherry et al., 1997; MacIntosh et al., 2000; Rodacki et al., 2001; Takaishi et al., 1998; Takaishi et al., 1996).

Numerous studies have found that increases in muscle activation via iEMG or RMS during incremental and constant-load exercise are associated with progressive

increases in  $\dot{V}O_2$  (Bull, Housh, Johnson, & Perry, 2000; Hug et al., 2004). The relationship between EMG and  $\dot{V}O_2$  is considered to occur due to an activation of additional muscle fibres required to compensate for decrements in force capacity caused by fatiguing fibres (Borrani, Candau, Perry, Millet, & Rouillon, 2003; Bull et al., 2000; Hug et al., 2004). In addition, it has been found that EMG may disproportionately increase compared to  $\dot{V}O_2$  during an incremental test (Hug et al., 2004; Hug, Laplaud et al., 2003). This disproportional increase in EMG is thought to coincide with either the second (Hug, Faucher, Kipson, & Jammes, 2003; Hug, Laplaud et al., 2003) or both (Lucia, Sanchez, Carvajal, & Chicharro, 1999) ventilatory thresholds. This is supported by Hug et al. (2004) who showed when examining vastus lateralis during a progressive exercise test that a greater RMS/ $\dot{V}O_2$  ratio was found in endurance-trained subjects, which is thought to be due to a greater percentage of slow oxidative fibres exhibited by well-trained cyclists (Coyle, Sidossis, Horowitz, & Beltz, 1992; Horowitz, Sidossis, & Coyle, 1994; Takaishi et al., 1998). However, as stated previously, whilst IEMG and RMS indicate overall motor unit recruitment, they fail to provide information regarding the type of fibre recruitment (i.e. Type I or Type II fibres) (Borrani et al., 2003; Hug, Laplaud et al., 2003). A further study examining EMG during incremental exercise found EMG threshold to occur at similar times for all muscles recorded (with differing fibre composition). This then suggests that motor unit recruitment is not altered by fibre type (Hug, Laplaud et al., 2003). How well this relates to prolonged constant-load or submaximal exercise is unknown.

The use of magnetic resonance imagery (MRI) has shown that during constant load cycling above threshold levels (60% of difference between lactate threshold and  $\dot{V}O_{2\max}$ ) a greater muscle mass is utilised compared to below threshold levels (Saunders et al., 2000). This suggests a greater activation of fast-twitch muscle fibres and consequently a progressively greater  $\dot{V}O_2$  and lactate accumulation (Ebenbichler et al., 2002; Green, 1997; Hamlin & Quigley, 2001b; Saunders et al., 2000). However, studies have shown that when subjects cycle at submaximal intensities (at or below ventilatory threshold) for prolonged periods (>two hours), no significant relationship is shown between whole body  $\dot{V}O_2$  and EMG of the vastus lateralis (Bull et al., 2000; Hug et al., 2004), or MRI transverse

relaxation times of the lower extremities during 15 minutes of cycling (Saunders et al., 2000). In these studies it is unclear as to the activation of synergistic and antagonistic muscles that have been previously been shown to respond accordingly in order to compensate the fatiguing agonistic muscles (Hautier et al., 2000; Hunter, St Clair Gibson, Lambert et al., 2002).

## **2.6.2 Stretch/shortening cycle**

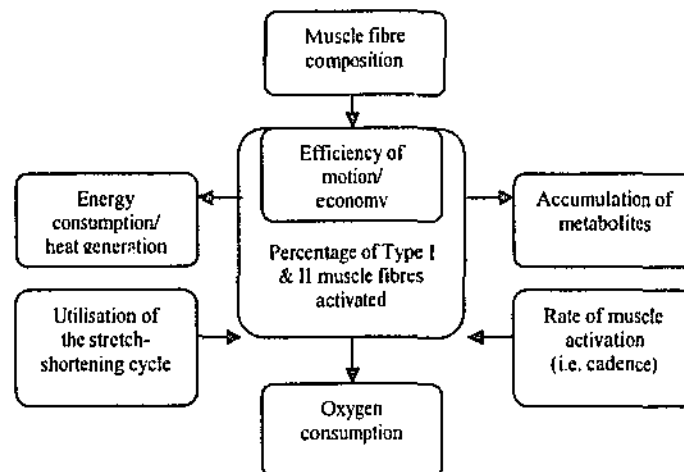
Cycling has long been considered to be an exercise involving solely concentric contractions, whereby patterns of eccentric contractions are thought to be minimal. However, recent research has found that during high pedal velocity, eccentric muscle contractions do take place (Gollhofer, Komi, Fujitsuka et al., 1987; Gollhofer, Komi, Miyashita et al., 1987; Komi, 2000; Nicol et al., 2003). This eccentric muscle action that occurs during the lengthening phase is then followed by a concentric (shortening) action (Gollhofer, Komi, Miyashita et al., 1987; Komi, 2000; Nicol et al., 2003). This combination of lengthening and shortening is known as the stretch-shortening cycle (Gollhofer, Komi, Miyashita et al., 1987; Komi, 2000). Komi (2000) has stated that during hopping, running and jumping exercises (where a majority of the research into stretch-shortening cycle has been focused), muscle-tendon lengthening can increase by 6-8%. It has been found that a portion of the stored elastic energy in ligaments and tendons can be recovered during the shortening phase of motion, producing an enhancement of force and power production (Gollhofer, Komi, Miyashita et al., 1987; Hausswirth et al., 2000; Komi, 2000; Noakes, 2000b), thus improving the potential economy of cycling. Stretch-shortening fatigue may also occur in response to a failure of the contractile capacity of a muscle occurring in response to muscle damage caused during the stretch-shortening cycle (Gollhofer, Komi, Fujitsuka et al., 1987; Gollhofer, Komi, Miyashita et al., 1987). Furthermore, the stretch-shortening cycle influences muscle mechanics, joint and muscle stiffness and reflex involvement, thus causing a diminution in the tolerance to muscle stretching and an increase in time to shift from muscle stretch to muscle contraction (Brooks et al., 2000b; Davis & Bailey, 1997; Kay & Marino, 2000). The increased contraction times during both eccentric and concentric phases, and the resultant reduction in force production during repetitive sub-maximal stretch-shortening cycles are considered to be a result of a decrease in



reflex components, which are interpreted as a protection mechanism of the central nervous system (Davis & Bailey, 1997). It is generally acknowledged that afferent feedback resulting from muscle tension is provided by the Golgi tendon organs (Cafarelli, 1982; Gregory, Brockett, Morgan, Whitehead, & Proske, 2002; Hutton & Nelson, 1986). It has been hypothesised that these sensations are causal for generalised sensations of muscular fatigue (Gregory et al., 2002; Hampson et al., 2001). However, a study involving eccentric exercises performed on the gastrocnemius muscle of an anaesthetised cat found that there were insignificant increases in tendon organ sensitivity (Gregory et al., 2002). It was then concluded that even when the muscles are fatigued, central factors relating to perception of effort play a significantly larger role than peripheral alterations in perception of force development.

### 2.6.3 Summary of the biomechanical model

The biomechanical model of fatigue (Figure 5) is predominantly based upon the idea that an enhanced efficiency of motion results in better economy. Thus, less demand is placed upon other physiological mechanisms that may be responsible for fatigue, causing reduced oxygen and energy consumption (see energy supply/energy depletion model), and a reduction in the development of intramuscular metabolites (see cardiovascular/anaerobic model) and core body temperature (see below).



**Figure 5.** The biomechanical model of fatigue states that improved mechanical efficiency and economy during cycling leads to a greater activation of slow twitch muscle fibres at a given workload. Improved efficiency and economy then reduces the demands placed on energy consumption and heat generation.

## 2.7 Thermoregulatory Model

The negative effects of environmental heat and hyperthermia on exercise performance have been well established (Gleeson, 1998; Hargreaves & Febbraio, 1998; Hunter, St Clair Gibson, Mbambo et al., 2002; Kay, Taafe, & Marino, 1999; Marino et al., 2000; Moran, 2001; Nielsen, 1998; Nielsen et al., 1993; Tucker et al., 2004). An increase in environmental temperature beyond a thermally neutral environment (such as 21° to 26°C) (Cheuvront & Haymes, 2001; Coyle & Montain, 1992; Hargreaves & Febbraio, 1998; Kay & Marino, 2000; Marino, Kay, Cannon, Serwach, & Hilder, 2002; Tatterson, Hahn, Martin, & Febbraio, 2000; Yoshida et al., 1997) creates a greater demand upon the body when exercising (Cheuvront & Haymes, 2001; Cochrane & Sleivert, 1999; Hunter, St Clair Gibson, Mbambo et al., 2002; Marino et al., 2000; Wilmore & Costill, 1999). Under such conditions, exercise performance in relation to time to exhaustion or work output is compromised (Cochrane & Sleivert, 1999; Coyle & Montain, 1992; Febbraio, 2000; Hargreaves & Febbraio, 1998; Kay et al., 1999; Marino et al., 2000; Wilmore & Costill, 1999). For example, Tatterson et al. (2000) showed that during a 30-min time trial, performed by well-trained cyclists, mean power output was reduced by 6.5% under hyperthermic (32°C) compared to thermally neutral (23°C) environments.

Heat created in the body during exercise is transferred to the environment via a number of differing methods (Febbraio, 2000; Watt, Garnham, Febbraio, & Hargreaves, 2000; Wilmore & Costill, 1999). Rises in core body temperature are affected by the rate of metabolic heat production (muscle metabolism) and heat removal/dissipation (convection, conduction, radiation or evaporation) (Gray & Nimmo, 2001; Kay et al., 1999). Without adequate heat removal/dissipation, exercising muscle producing heat results in a progressive increase in core body temperature (Febbraio, 2000; Hargreaves & Febbraio, 1998). Metabolic heat production during exercise can increase core body temperature by 1°C every 5-7 min (Armstrong & Maresh, 1998; Cheuvront & Haymes, 2001; Coyle & Montain, 1992; Duffield, Dawson, Bishop, Fitzsimons, & Lawrence, 2003; Noakes, 2000b). However, core temperatures in excess of 40°C cannot be tolerated for prolonged periods (Cheuvront & Haymes, 2001; Nielsen, Strange, Christensen, Warberg, & Saltin, 1997; Noakes, 2000b). Thus, it is theorised that as the body reaches critical

core body temperatures (i.e.,  $\sim 40^{\circ}\text{C}$ ), exercise is limited by the production and dissipation of heat (Armstrong & Maresh, 1998; Hunter, St Clair Gibson, Mbambo et al., 2002; Nielsen et al., 1997).

### 2.7.1 Central thermoregulation fatigue theory

The hypothalamus receives afferent signals from peripheral thermal receptors, which in turn regulate central neural drive and sympathetic stimulation processes responsible for heat removal (i.e., sweat rate, peripheral blood flow) during exercise (Armada-da-Silva, Woods, & Jones, 2004; Hunter, St Clair Gibson, Mbambo et al., 2002; 2001). In a recent study, Nybo and Nielsen (2001) electrically stimulated the femoral nerve of endurance-trained cyclists during sustained (2-min) isometric contractions in a hyperthermic state ( $40^{\circ}\text{C}$ ) and found that reductions in maximal isometric force were the result of central rather than peripheral factors. This suggests that hyperthermic-induced fatigue may cause alterations that affect the ability of the CNS to supply a constant neural drive (Armstrong & Maresh, 1998; Nybo & Nielsen, 2001a, 2001c; Tucker et al., 2004). It has been hypothesised, therefore, that increases in core body temperature to 'critical' levels may cause a reduction in the rate of central activation (Cheuvront & Haymes, 2001; Kay & Marino, 2000; McArdle et al., 2001; Nybo & Nielsen, 2001a; Tucker et al., 2004; Wilmore & Costill, 1999). However, there is strong support to show that brain temperature is not affected under these temperatures (Nybo & Nielsen, 2001b).

Evidence for the central thermoregulatory fatigue model stems from a classic study performed by Nielsen et al. (1993), who found that when well-trained cyclists cycled to exhaustion ( $60\% \dot{V}\text{O}_{2\text{max}}$ ) in hot  $40\text{--}42^{\circ}\text{C}$  conditions, exercise was terminated when core body temperature (oesophageal) reached approximately  $39.5^{\circ}\text{C}$ . Reductions in muscle or skin blood flow, accumulation of metabolites (i.e.,  $\text{K}^+$ , lactate), or a lack of substrates (blood glucose and free fatty acids) were not observed. Thus, increases in core body temperature to 'critical levels' was ruled as the cause of fatigue (Nielsen et al., 1993). More recently, however, Tucker et al. (2004) have shown that rectal temperatures during a 20-km cycling time trial in amateur cyclists were not significantly different between hot ( $35^{\circ}\text{C}$ ) and cool ( $15^{\circ}\text{C}$ ) conditions, except in the final recording (20-km), where rectal temperatures were  $39.2^{\circ}\text{C}$  and  $38.8^{\circ}\text{C}$ , for hot and cool conditions, respectively. Nevertheless, power

output appeared to decline in the hot climate after only 30% of the total duration (6-km) and was significantly different from the cool environment after 80% of the total duration (~219 vs. 260W, for hot vs. cool conditions, respectively), suggesting that decrements in power output were not solely the result of an increase in core body temperature. This is supported by the fact that the highest power output in both trials occurred when core body temperature was greatest (39.2°C and 38.8°C during the final 5% of the time trial). Furthermore, IEMG was shown to be significantly reduced in the hot condition at 10-km of the trial when core body temperatures were not significantly different. It was therefore proposed that reductions in IEMG and power output were the result of a 'cerebral anticipatory response', used to maintain a homeostatic body temperature (Tucker et al., 2004).

During prolonged submaximal cycling, the onset of hyperthermia has been shown to be associated with reductions in cerebral circulation (Nybo & Nielsen, 2001b) due to increases in ventilation (hyperventilation) under hyperthermic conditions. This causes a decrease in arterial carbon dioxide pressure (Kayser, 2003; Nybo & Nielsen, 2001b), which in turn has a significant effect on blood flow to the brain (Nybo, Moller, Volianitis, Nielsen, & Secher, 2002). This reduction in brain blood flow may consequently result in a decreased substrate (oxygen and ATP) supply and waste product removal, to and from the brain (Gray & Nimmo, 2001; Kay et al., 2001). The central thermoregulatory fatigue model therefore states that prolonged exercise performance is limited by hyperthermic-induced alterations in brain activity as opposed to peripheral factors associated with muscle activation. An extension of this model was postulated by Tatterson et al. (2000), whom stated that brain activity is sensitive to increased arterial blood temperatures, and is thus related to core body temperature. Indeed, it has also been shown that increases in core body temperature, and especially muscle temperature, results in an increased contraction velocity and contraction speed, leading to an earlier and more pronounced neuromuscular fatigue (Armstrong & Maresh, 1998; Hunter, St Clair Gibson, Mbambo et al., 2002; Wilmore & Costill, 1999).

### **2.7.2 Peripheral thermoregulation fatigue theory**

Increases in skin temperature and the onset of hyperthermia is suggested to be responsible for a reduction in the “will” or “drive” for performance (Hunter, St Clair Gibson, Mbambo et al., 2002; Nielsen, Hyldig, Bidstrup, Gonzalez-Alonso, & Christoffersen, 2001; Nybo & Nielsen, 2001b, 2001c). During prolonged exercise in elevated thermal environments, increases in rating of perceived exertion parallel increases in core body temperature (Hunter, St Clair Gibson, Mbambo et al., 2002; Nybo et al., 2002; Nybo & Nielsen, 2001b). On the contrary, Tucker et al. (2004) showed no significant difference between rating of perceived exertion and power output or IEMG in hot versus cool climates. This implies that the reduced neural drive (EMG) is in fact the result of centrally controlled alterations to muscle recruitment, which might be influenced by elevated skin and muscle temperatures (Ray & Gracey, 1997; Tucker et al., 2004). However, limitations exist in much of the recent research involving thermoregulation and EMG, in that other psychological markers that have the potential to influence performance (i.e., thermal sensation and muscle pain) are often not measured (Armada-da-Silva et al., 2004). Interestingly, Armada-da-Silva et al. (2004) found that after passive heating during a fatiguing cycle protocol, face cooling (via a mist fan) significantly reduced the ratings of perceived exertion of participants compared to a control group, irrespective of similar perceptions of thermal comfort.

It is considered that the more efficient one can be at a given power output or speed, the lower the oxygen consumption and heat production there will be, which should in theory increase fatigue-resistance (Tucker et al., 2004). This is supported by Marino and colleagues, (Marino et al., 2000) who showed a positive correlation ( $r=0.74$ ,  $P<0.001$ ) between heat storage and body mass during an 8-km run in thermally elevated conditions (35°C); no relationship was found to exist at 25°C or 15°C.

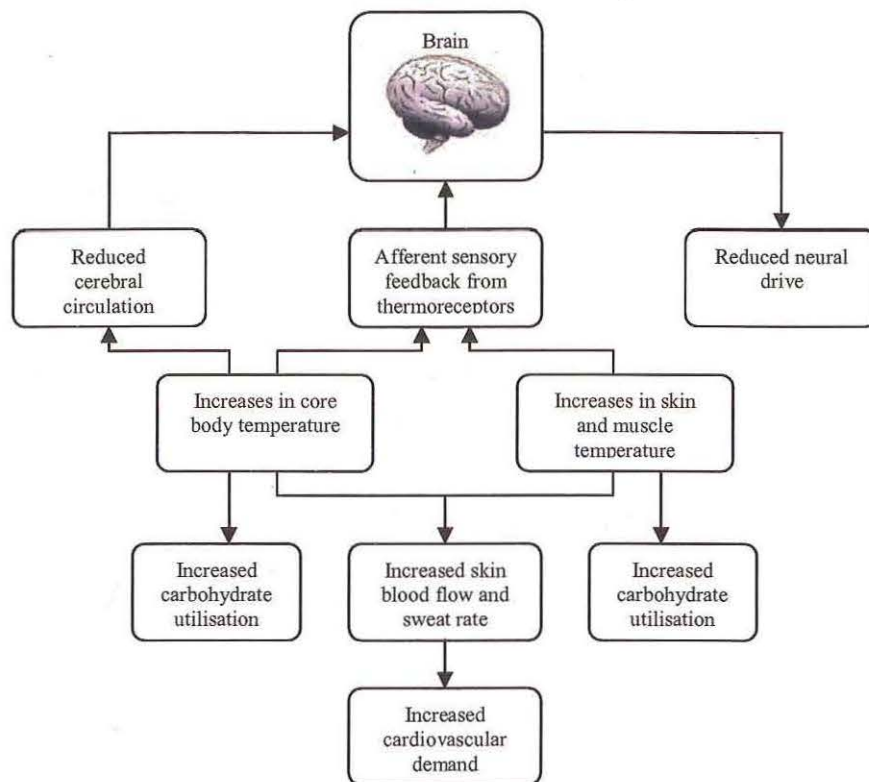
An increase in the body temperature is sensed by the thermoreceptors located in the hypothalamus (Cheuvront & Haymes, 2001; Cochrane & Sleivert, 1999; Gray & Nimmo, 2001; Hargreaves & Febbraio, 1998; Tikuisis et al., 2002; Wilmore & Costill, 1999), causing a reflex response initiating an increase in skin blood flow and sweating rate (Cochrane & Sleivert, 1999; Green, 1997; Nielsen et al., 1993; Nybo & Nielsen, 2001b). This increases the demand placed upon the cardiovascular system as

it must not only supply blood to working muscles, but it must also shunt systemic blood flow to the skin to dissipate heat (Hunter, St Clair Gibson, Mbambo et al., 2002; Nybo, Jensen, Nielsen, & Gonzalez-Alonso, 2001). Thus, it is believed that during exercise under thermally elevated conditions, cardiac output may remain constant or in fact be reduced (Nybo & Nielsen, 2001b) due to the onset of hyperthermia and dehydration (Gray & Nimmo, 2001; Hunter, St Clair Gibson, Mbambo et al., 2002). Increases in skin blood flow may also cause a reduction in splanchnic, renal (Nybo et al., 2001; Nybo & Nielsen, 2001b) and in some conditions muscle blood flow (Hunter, St Clair Gibson, Mbambo et al., 2002; 1998). Interestingly, Nielsen et al. (1997) showed that when cycling at 45%  $\dot{V}O_{2max}$ , a plateau in skin blood flow (determined via venous occlusion plethysmography and laser doppler flowmetry) occurred despite continuing rises in core body temperature. If skin blood flow reaches a limit during exercise, due to the demands of working muscle and vital organs (i.e. heart and brain), this may have a significant effect on core body temperature and thus fatigue. Therefore temperature regulation during exercise is directly linked to the cardiovascular/anaerobic model of fatigue (Brooks et al., 2000b; Gray & Nimmo, 2001; Gray, Devito, & Nimmo, 2002; Hunter, St Clair Gibson, Mbambo et al., 2002; McArdle et al., 2001; Wilmore & Costill, 1999).

Increases in core body and skeletal muscle temperatures have also been associated with an increase in carbohydrate utilisation (Armada-da-Silva et al., 2004; Gray & Nimmo, 2001; Kay & Marino, 2000), highlighted by increased muscle lactate accumulation, gluconeogenesis, increase liver glucose output, and increased blood and glycogen oxidation (Armada-da-Silva et al., 2004; del Aguila et al., 1999). Parkin et al. (1999) have shown that during a fatigue-cycling protocol (70% of  $\dot{V}O_{2peak}$  until exhaustion) the glycogen content of the vastus lateralis in endurance-trained men was higher under a thermally elevated environment (40°C) compared to normal (20°C) or cold temperatures (3°C). This increase in carbohydrate utilisation is considered to be the result of a heat-induced stimulus for muscle gluconeogenesis, consequently placing greater demands upon glycogen stores (Gleeson, Eston, Marginson, & McHugh, 2003; Green, 1997).

### 2.7.3 Summary of the thermoregulatory model

The thermoregulatory model of fatigue suggests that a critical core body temperature may exist whereby upon attainment of this temperature, exercise is reduced or terminated. The increase in core body, muscle and skin temperature also causes increased demands to be placed on other physiological systems/models that may be responsible for fatigue during prolonged cycling (Figure 6). These include the cardiovascular/anaerobic model, the neuromuscular model, the energy supply/energy depletion model, and the psychological model.



**Figure 6.** The thermoregulatory model of fatigue states that fatigue is a result of the body reaching critical levels of temperature in either the core, muscle and/or skin. This creates an increase in skin blood flow and a resulting increase in cardiovascular demand. Heightened temperatures may also lead to a reduced neural drive due to afferent sensory feedback from both central and peripheral thermoreceptors.

## **2.8 Psychological/Motivational Model**

The psychological/motivational model of fatigue can be defined as a lack of enthusiasm or interest in exercise performance (Brooks et al., 2000b; Febbraio, 2000; Hargreaves & Febbraio, 1998; Nybo & Nielsen, 2001a; Tattersson et al., 2000), and is often incorporated as a part of the neuromuscular model of fatigue (Armstrong & Maresh, 1998; Brooks et al., 2000b; Cafarelli, 1982; Kayser, 2003; Tattersson et al., 2000). As addressed by Noakes (2000), the central fatigue model (see below) suggests that a reduction in performance, and thus fatigue, occurs on a subconscious level to avoid damage or death occurring during exercise. However, the psychological/motivational model holds that neuromuscular function is intentionally altered, thus causing a decrease in motor control activation (Borg, 1982; Cain & Stevens, 1973; Cochrane & Sleivert, 1999; Kayser, 2003; Tattersson et al., 2000).

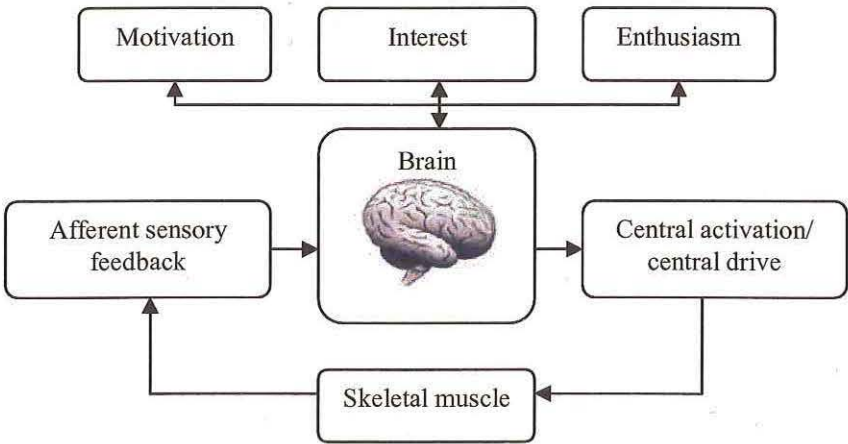
To date, it is unclear as to the precise mechanism that effects the brain's response to afferent feedback during exercise (Armada-da-Silva et al., 2004; Hampson et al., 2001). It is thought that numerous factors, including muscle trauma, skin temperature, blood lactate (Hampson et al., 2001), heart rate, respiratory rate, exercise task, minute ventilation, oxygen uptake (Hampson et al., 2001; Skinner, Hutsler, Bergsteinova, & Buskirk, 1973), and mode of exercise (Brooks et al., 2000b; Hampson et al., 2001; Laursen, Shing, & Jenkins, 2003b) (i.e., cadence (Gotshall et al., 1996; Pandolf & Noble, 1973) may have a psychological influence on performance. Yet, it is unclear as to the specific effect that each one of these variables has on physiological fatigue. To better understand the relationship between psychological influences and exercise performance a number of models have been created, with one of the most widely accepted being Borg's 15-point rating of perceived exertion (RPE) scale (Borg, 1982). From research using this, and other models, it has been suggested that the onset of acute and chronic fatigue is associated with increases in RPE (Halsen et al., 2002; Ulmer, 1996). It has been found that RPE has a greater relationship with skin temperature and heart rate, as opposed to changes in core body temperature (Armada-da-Silva et al., 2004; Brooks et al., 2000b). Alternately, a greater RPE has been associated with increases in ventilation rate and minute ventilation (Hampson et al., 2001). Moreover, when exercise involves a 'closed-loop' design, whereby subjects are aware of the overall distance to be



completed, subjects can significantly reduce their trial by trial repeatability (Laursen, Shing, & Jenkins, 2003c), compared to an open-loop time to exhaustion test (Armstrong & Maresh, 1998; Green, 1997). It has been suggested by Ulmer (1996) that subjects anticipate the work required for a task and alter power output as to complete the activity with the best possible performance but avoid fatigue. This is supported in findings which show that the lower the duration of professional cycling competitions the greater the intensity tends to be (Lucia, Hoyos, Santalla, Earnest et al., 2003).

**2.8.1 Summary of the psychological/motivational model**

Collectively, the literature has shown that, to date, there is no apparent single physiological variable responsible for motor output alteration from afferent signals (Figure 7). From this, it is assumed that numerous mechanisms are responsible for psychological alterations in central activation and perceived exertion (Hampson et al., 2001), which in turn determine the onset of fatigue, reductions in cycling power output and ultimately the point of exhaustion during prolonged cycling.



**Figure 7.** The psychological/motivational model states that central drive is reduced due to lower motivation, interest and/or enthusiasm for the exercise task. The reduced enthusiasm may or may not be related to afferent sensory feedback.

## 2.9 Central Governor Model

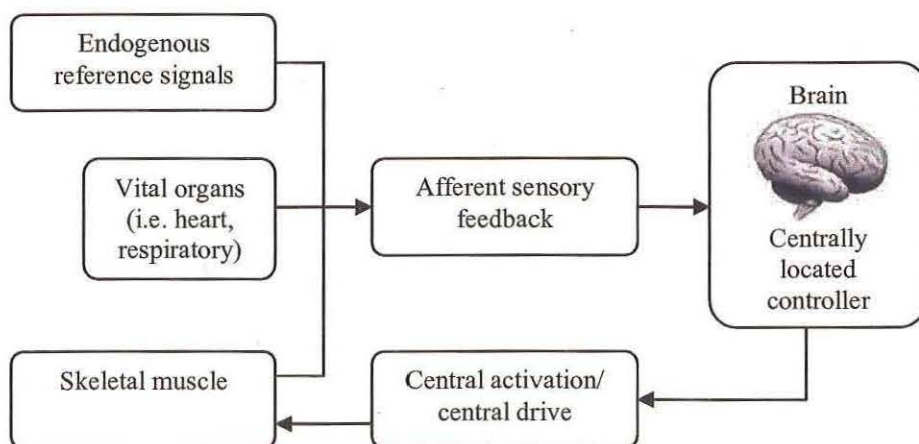
Noakes' (2001) central governor model is based on the idea that skeletal muscle activation is controlled by a regulator located either in the heart, the brain, or along the neuromuscular pathway, in order to protect vital organs from injury or damage (Hampson et al., 2001; Lucia, Hoyos, Santalla, Earnest et al., 2003). Relationships have been found to exist between cardiac output and skeletal muscle performance (Brooks et al., 2000a, 2000b; de Vries & Housh, 1994; Faulkner et al., 1977). However, as recognised by Noakes (2000b), if cardiac output does in fact limit performance, then activation of skeletal muscle must be reduced prior to the heart reaching its maximum output, in order to protect blood flow and therefore vital organs (heart, brain and respiratory muscles) (Hampson et al., 2001; Noakes, 2000b; Noakes et al., 2001). Noakes states that if a maximum cardiac output is reached during exercise, this would cause a limit in coronary blood flow, and as the heart relies upon its own blood flow, the capacity of the heart to pump blood would be effected, leading to myocardial ischaemia (Noakes, 2000b; Noakes et al., 2001). Evidence of such a protective mechanism may explain why during maximal exercise, cardiac output, muscle recruitment, and thus exercise performance is reduced in hypoxic conditions (i.e., altitude) (Hahn & Gore, 2001; Hampson et al., 2001; Noakes, 2000b). Recent research into the effects of a hypoxic environment on the heart during submaximal exercise found that an 80-km cycle time trial did not produce any exercise-induced cardiac fatigue or significant cardiac damage (no significant increase in troponin-T) (Shave et al., 2004). In relation to heart rate and performance times, considerable differences were not found between hypoxic and normoxic environments (Shave et al., 2004). The fact that no cardiac damage was found in this study suggests that a central regulator may have reduced neural drive in order to protect the heart. However, this can not be confirmed, as performance times were similar and muscle activation was not determined. Moreover, Ide et al. (1998) showed that along with muscle blood flow, cerebral circulation velocity is compromised by insufficient cardiac output during exercise involving a large muscle mass (as in cycling). This suggests that if exercise performance is controlled via a central governor, as suggested by Noakes et al., (2001) then reductions in cardiac output may affect its activity.

What is more, it has been suggested that a central regulator is also active during submaximal exercise, whereby a central programmer regulates power output based upon anticipated exercise requirements (Hampson et al., 2001; Lucia, Hoyos, Santalla, Earnest et al., 2003; Tucker et al., 2004; Ulmer, 1996). It has thus been hypothesised that during three-week tour races (i.e., Tour de France and Vuelta a España) the inverse relationship that exists between exercise volume and intensity is reflective of a “teleoanticipatory” response activated via the central nervous system (Lucia, Hoyos, Santalla, Earnest et al., 2003; Ulmer, 1996), whereby a subconscious increase in intensity occurs when volume (i.e., distance) is less. The activation of this teleoanticipatory response is considered to be due to afferent sensory input, causing a reduced central activation in order to maintain physiological homeostasis (Hampson et al., 2001). Further support for the existence of a teleoanticipatory system can be seen in a recent study by Kay et al. (2001), who found that during a 60-min cycle protocol, where 1-min all-out sprints were held every 10-min, subjects were able to increase neural drive (EMG) to restore power output in the final sprint back to values obtained in the first sprint, indicating the presence of a neural reserve. This increase in EMG activity may represent psychological factors to exercise performance (i.e. pacing strategies), as subjects were able to monitor the course profile, observing the occurrence of sprints.

In an attempt to better understand the effects of perceived exertion upon central command, Williamson et al. (2001) hypnotically manipulated effort sense of six highly hypnotisable individuals to determine alterations in cerebral activation. From this study it was found that hypnotically-increased effort sense during constant-load exercise could increase blood pressure, heart rate, as well as activation of the right insular cortex and right thalamic regions of the brain (Williamson et al., 2001). It was then suggested that as hypnotically-reducing sense of effort failed to reduce heart rate or blood pressure, then afferent sensory information from skeletal muscle must play a role in the magnitude of cardiac activation as found in the central governor model (Williamson et al., 2001). This study is, however, limited in as much as neither muscle activation nor afferent firing rates were determined (Williamson et al., 2001).

During acute and chronic hypoxia, the reduction in heart rate, stroke volume and cardiac output are harmonious with the theory of a central controlling governor.

This theory is supported by previously stated theories (Central Activation Failure Theory and Psychological/Motivational Model), which holds that skeletal muscle activation is controlled by the brain (Figure 9). Therefore, as maximal exercise performance is thought to include the central nervous system, studies in fatigue should assess neuromuscular function/dysfunction in an attempt to determine if such a governor exists. Furthermore, to our knowledge studies attempting to determine the actual involvement of a central governor during cycling at both maximal and sub-maximal intensities has yet to be performed.



**Figure 8.** The central governor model states that efferent drive is controlled in response to afferent sensory feedback. Central activation is then limited in order to protect vital organs against ischemia or tissue death.

## 2.10 Summary and Conclusion

Much of the previous research into fatigue has found that cycling performance can be limited by a number of differing mechanisms that have been proposed in numerous models (Figure 9). These models include the cardiovascular/anaerobic model, energy supply/energy depletion model, the neuromuscular fatigue model, the muscle trauma model, the biomechanical model, the thermoregulatory model, psychological/motivational model, and the central governor model. These models provided evidence that fatigue during prolonged cycling may occur in relation to a number of differing mechanisms (Gundersen, 1998) and the precise involvement of these models are dependant on the task performed (Hunter et al., 2004).

At maximal aerobic power output, cycling performance may be limited by the ability of the cardiovascular system to supply sufficient oxygen to the working muscle. However, at submaximal levels, fatigue may be due to neurological alterations causing a reduction in central drive. In fact, the available research suggests that during fatiguing exercises, more than one of these models may be applicable, and indeed, many of these models may be interrelated (Figure 9). Physical performance during cycling depends upon the condition of several systems of the body, including the nervous system, respiratory system, cardiovascular system and skeletal muscle (Gundersen, 1998). Clearly, further research examining more than one of these fatigue models concurrently is required to make clarity out of the numerous interrelating factors. This type of research will lead Sport Scientists to an improved understanding of the precise mechanisms that are associated with fatigue during cycling, and provide coaches and athletes with a greater insight on how to further improve cycling performance.



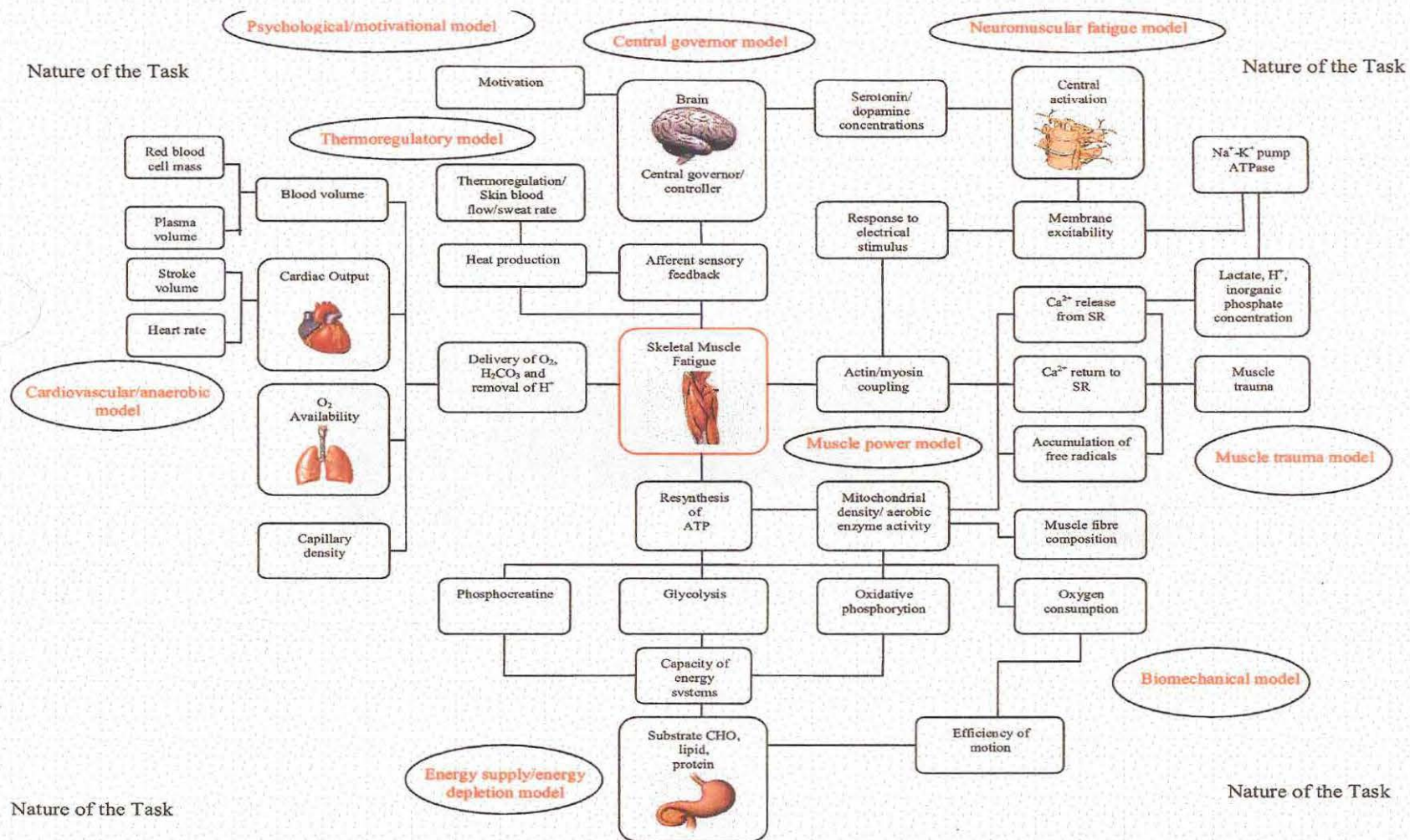


Figure 9. Relationship between fatigue models.

## **CHAPTER 3      METHODOLOGY**

### **3.1      Subjects**

Nine endurance-trained, male cyclists aged between 18 and 41 years volunteered to perform in this study (Appendix A). Subjects had at least one year of cycling experience, and were cycling between 300 and 600 km·wk<sup>-1</sup> (397 ± 93 km·wk<sup>-1</sup>) at the time of the investigation. To ensure a relatively homogenous group, subjects who recorded a  $\dot{V}O_{2\max}$  score of lower than 55 ml·kg<sup>-1</sup>·min<sup>-1</sup> during the initial testing session were excluded from the study.

### **3.2      Experimental Protocol**

All tests were performed in the exercise physiology climate chamber at the School of Biomedical and Sports Science, Edith Cowan University (Joondalup, WA). Subjects reported to the laboratory on five separate occasions, spaced at least seven days apart, and performed all tests at the same time of day. Subjects were required to abstain from heavy training 24 h prior to testing.

In order to ensure that subjects maintained normal training commitments, subjects completed a training diary for the week prior to each test (Appendix B). Diet records were also completed 24 h prior to testing to control for carbohydrate intake, and subjects were educated and encouraged to make appropriate food choices during this period (Appendix C). Subjects consumed at least 6 g of carbohydrate per kg of body weight on the day prior to, and at least 1 g of carbohydrate per kg of body weight on the morning of each trial (St Clair Gibson, Schabort et al., 2001).

Subjects were informed as to the experimental protocol and hypotheses at the time of recruitment (Appendix D). All subjects signed a document of informed consent (Appendix E) and a medical questionnaire (Appendix F) and the study protocol was performed similar to the methods of previously published studies that examine fatigue during cycling (St Clair Gibson, Schabort et al., 2001). The

experimental procedure was approved by the Edith Cowan University Central Human Research Ethics Office prior to commencement.

### **3.2.1 Testing Schedule**

A general overview of the testing sessions (all completed approximately one week apart) is as follows:

#### Session 1:

- $\dot{V}O_{2max}$  familiarisation test
- Baseline measures (height and weight)

#### Session 2:

- Skinfolds
- $\dot{V}O_{2max}$  / anaerobic threshold test

#### Session 3 - Familiarisation:

- Multidimensional fatigue inventory (MFI-20)
- Reaction time test (IT/RT)
- Familiarisation of procedures to obtain maximal EMG activity for normalisation purposes
- Familiarisation 100-km time trial at 22°C and 40% relative humidity

#### Session 4 and 5:

- Multidimensional fatigue inventory (MFI-20)
- Reaction time test (IT/RT)
- Determination of maximum voluntary isometric contraction (MVIC) for EMG normalisation
- 100-km time trial at either 10°C or 34°C (randomly selected) and 40% relative humidity



### 3.3 Data Collection Procedures

#### 3.3.1 Subject characteristics

##### 3.3.1.1 $\dot{V}O_{2\max}$ test

$\dot{V}O_{2\max}$  was determined during an incremental exercise test to exhaustion using the Velotron Cycle Ergometer and Velotron Coaching Software (Version 1.5) (RacerMate; Seattle, WA, USA). This test began with subjects cycling at a power output of 100 W for 5 min. This power output then increased at a rate of 50 W every 5 min. Subjects were allowed to alter their pedal rate to their preferred cadence as the Velotron ergometer alters resistance based on cadence. The test was terminated however, when subjects were no longer able to maintain a cadence of at least 60 rev·min<sup>-1</sup> (Kay et al., 2001; Marino et al., 2002; St Clair Gibson, Schabort et al., 2001). Peak power output (PPO) was recorded as the highest stage completed. If the subject finished part way through a 5-min stage, PPO was pro-rated. E.g., If a subject finished 2 min 30 s into the 300 W stage, their PPO would be: [(2.5 min/5 min) x 50] + 300 = 325 W (Hawley & Noakes, 1992).

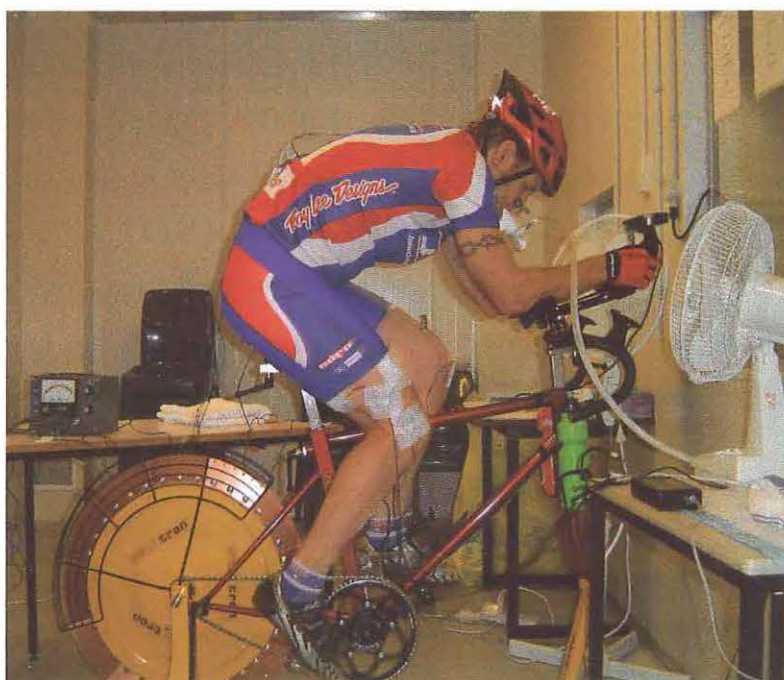
Inspired and expired air was measured throughout the entire test using the Miedgraphics CPX Gas Analysis System (St. Paul, MN, USA). The gas analyser was calibrated immediately prior to all tests with alpha gases, and the ventilometer was calibrated using a syringe of a known volume.  $\dot{V}O_{2\max}$  was defined as the average of the two highest  $\dot{V}O_2$  values over an averaged 15-s period (Laursen et al., 2003b). Subject's RPE (Borg, 1982) (Appendix G), pain intensity in the thigh (quadriceps) (Micalos, Marino, & Kay, 2004), thermal sensation (Appendix H) (Young, Sawka, Epstein, Decristofano, & Pandolf, 1987) (Appendix I) and blood lactate using the i-STAT portable blood gas analyser (i-STAT Corporation, East Windsor, NJ, USA) was recorded at the end of each workload. Heart rate was automatically recorded at 5-s intervals using a heart rate monitor (S710i Polar; Polar Electro Oy™; HQ, Kempele, Finland). At least seven days before this test, subjects performed a familiarisation test using the same procedures outlined, minus the blood lactate measures.

### 3.3.1.2 Baseline measures

During the second testing session, baseline measures of height, body mass, resting heart rate, and the sum of nine skinfolds were recorded. Height was measured to the nearest 0.5 centimetre with a Seca stadiometer (Brooklyn, NY), and body mass was measured with Mettler Toltdo ID1 scales to the nearest 10 grams (Columbus, OH, USA). Resting heart rate was recorded after subjects sat quietly on a chair for a period of 5 min, under standard laboratory conditions ( $\sim 22^{\circ}\text{C}$ ). Skinfolds were measured in duplicate from nine sites (biceps, triceps, subscapular, pectoral, mid-axilla, supraspinale, mid-abdominal, mid-thigh and medial calf) by the same researcher using Harpenden skinfold callipers (British Indicators Ltd, UK), with the mean value used to calculate total skinfolds.

### 3.3.2 Bike set up and determination of power output

Each cycling test was performed on the Velotron cycle ergometer, with the bike adjusted so as to replicate the habitual positioning of subjects on their own bicycle. Aerobars were also fitted to the ergometer to further replicate the subject's customary riding posture. The ergometer was also equipped with the subject's pedals, which allowed subjects to wear their own cycling shoes (cleats). On some occasions, the subjects' own saddle (seat) was fitted to the ergometer, depending on perceived comfort during the initial  $\dot{V}\text{O}_{2\text{max}}$  test. Each subject used the same saddle during all time-trials.



**Figure 10.** Example of subject positioning and set up during the 100-km time trials.

Following the  $\dot{V}O_{2\max}$  test, subjects performed, on separate days spaced at least seven days apart, three 100-km self-paced time-trials in different temperatures (thermally neutral 22°C, hot, 34°C and cold, 10°C), with a common relative humidity of 40%. The first test was a familiarisation test completed at 22°C, followed by a randomised crossover design of the warm and cold conditions; four subjects completed the cold trial first, while five subjects completed the hot trial first. Using the results from the progressive exercise test, each subject was prescribed a standardised 10-min warm-up consisting of 3 min at 25% PPO, 5 min at 60% PPO, and 2 min at 80% PPO. This warm-up protocol was a modified version of a warm-up typically used by professional cyclists before the completion of time-trials (Dr. David T. Martin, personal communications). Exactly 5 min following the completion of the warm-up, subjects began the 100-km time trial. Subjects aimed to complete the trial in the shortest possible time.

In an attempt to mimic the dynamic characteristics of cycle racing, to avoid a decline in performance due to monotony, and also provide a further measure of performance, subjects performed a number of high-intensity epochs during each of the time trials. In total, five all-out, 1-km sprints at 10, 32, 52, 72, & 99-km as well as four 4-km sprints at 20, 40, 60 & 80-km were performed. Commencement of these sprints was displayed on a computer and developed with the use of the Velotron 3D Software (Version 1.0; RacerMate, Seattle, WA) and cyclists were also instructed to complete the sprints in the shortest possible time. Completion of each sprint was displayed on the screen using the Velotron software. Subjects were requested to remain in a seated position throughout the sprints. A similar test to this has been validated by St Clair Gibson et al. (2001) who reported the between-test correlation coefficient to be 0.93 [95% confidence interval (CI), 0.79-0.98], and within-subject coefficient of variation to be 1.7% (95% CI, 1.1-2.5%).

During the time-trials, subjects were allowed to alter their gear ratio and pedalling cadence as required. Throughout the time-trials, power output, cycling speed, and performance times were automatically recorded via the Velotron 3D software. For analytical purposes, data from the Velotron 3D software was sampled every second. Power output was then time normalised by averaging each of the sprint and non-sprint stages. These averages were then normalised to the subjects' PPO determined during the  $\dot{V}O_{2\max}$  test.

### 3.3.3 Physiological variables

#### 3.3.3.1 Determination of skin, rectal and mean body temperature

To better represent competition cycling and realistic head and skin temperatures, subjects wore their own helmets and the same clothes during each of the time-trials. Once the subject was seated on the bike, resting measures of rectal ( $T_{rec}$ ) and skin ( $T_{skin}$ ) temperature (5 sites) were taken. These variables were again recorded three minutes following the warm up.  $T_{rec}$  and  $T_{skin}$  were also recorded at 19 different distances (0, 5, 10.5, 15, 22, 28, 32.5, 36.5, 42, 48, 52.5, 56.5, 62, 68, 72.5, 76.5, 82, 88, 95, and 100-km) during the time trials. Skin temperatures were measured using flat top copper skin thermistors (6.2 cm in diameter). Five skin thermistors (YTS Temperature, 400 Series; Dayton, OH) were attached on the chest, arm, thigh, calf, and forehead as follows:

- Chest – 1 cm inferior to the right axilla, between the anterior fold and the nipple;
- Arm – the midline of the anterior aspect of the right arm at a point midway between the acromion process and the olecranon process;
- Thigh – midline of the right anterior thigh midway between the inguinal crease and the proximal border of the patella;
- Calf – the midline of the medial aspect of the lower right leg at the level of maximal calf girth;
- Forehead – 3 cm superior to the superior border of the nasal bone

(Lepers et al., 2002; St Clair Gibson, Schabort et al., 2001; Tucker et al., 2004).

To insure that the thermistors remained in the desired position, Fixomull™ was placed over the sensors. Furthermore, to ensure that the subject's normal cycling pattern was not interrupted, the wires from the skin thermistors were taped to the body and the bicycle using Fixomull™ to avoid extraneous movement of the wires. Mean skin temperature was determined by Ramanathan's formula:

$$T_{skin} = (0.3 \times T_{chest}) + (0.3 \times T_{bicep}) + (0.2 \times T_{thigh}) + (0.2 \times T_{calf})$$

(Cochrane & Sleivert, 1999)

To determine core body temperature, a sterile disposable rectal thermistor (Monatherm Thermistor, 400 Series; Mallinckrodt Medical, St. Louis, MO, USA) was self-inserted at a 12 cm depth from the sphincter. Subjects were given full detailed instruction on the proper insertion method. Subjects then self-inserted the probe in the change room prior to each time trial and after performing the MVC measurements.

Mean body temperature was determined by Burton's formula:

$$T_{\text{body}} = (0.65 \times T_{\text{rec}}) + (0.35 \times T_{\text{skin}})$$

(Cochrane & Sleivert, 1999)

In order to mimic environmental conditions, during all time trials, two fans were placed in front of the subject at 45° angles to the subject (see Figure 10), each providing a wind velocity of ~10 km·h<sup>-1</sup>. Both fans were started upon commencement of the warm-up.

### 3.3.3.2 Cardio-respiratory measures

Throughout the time trials,  $\dot{V}O_2$  was measured breath-by-breath (Medgraphics CPX Gas Analysis System) for a period of 2-min at ten non-sprint points (0, 5, 15, 28, 36.5, 48, 56.5, 68, 76.5, 88-km and the final 1500 m). During the time-trials, heart rate was automatically recorded every 15-s (S710i Polar heart rate monitor). The heart rate was averaged over each sprint and non-sprint period, and normalised to the maximum heart rate reached during the  $\dot{V}O_{2\text{max}}$  test.

### 3.3.4 Biochemical variables

#### 3.3.4.1 Blood analysis

During each blood sample, two 125 µL electrolyte-balanced heparinised capillary tubes (Clinitubes, Reflotron, Copenhagen, Denmark) were filled from a single finger-prick of the subject's fingertip (Unistik<sup>®</sup> 2 Extra; Owen Mumford Ltd, Oxford, UK). All blood samples were analysed outside of the climate chamber in a controlled laboratory condition of 22°C. The first of the filled capillary tubes was used for the 6+ i-Stat cartridge, while the other being used to fill the GC4+ i-Stat cartridge. Using the i-Stat Blood Gas Analyser (i-STAT Corporation, East Windsor,

NJ, USA), the i-STAT 6+ cartridge measured the concentrations of sodium ( $\text{Na}^+$ ), potassium ( $\text{K}^+$ ), chloride ( $\text{Cl}^-$ ), urea nitrogen, glucose, hematocrit (Hct), and calculated hemoglobin (Hb), while the GC4+ i-STAT cartridge measured the variables of pH,  $\text{PCO}_2$ ,  $\text{PO}_2$ , lactate and calculated base excess ( $\text{BE}_{\text{ccf}}$ ), bicarbonate ( $\text{HCO}_3^-$ ), total carbon dioxide ( $\text{TCO}_2$ ) and oxygen saturation ( $\text{sO}_2$ ). The i-STAT runs all its assays at 37°C. Once the subject was seated on the bike, a blood sample was taken. Blood samples were again taken three minutes following the warm up (see Appendix J for protocol). During the time trials, capillary blood samples were taken at 0, 28, 48, 68, 88 and 100-km

### **3.3.5 Biomechanical variables**

#### **3.3.5.1 Generation of maximal voluntary isometric contraction (MVIC)**

In order to elicit maximal voluntary isometric contraction (MVIC) for the normalisation of electromyography (EMG) related variables, subjects MVIC's for the hamstring, quadriceps and calf on their right leg using a Cybex II+ isokinetic dynamometer (Huntsville, AL). EMG normalisation procedures were completed prior to each 100-km performance time trial. For the quadriceps and hamstring MVIC, trunk-thigh angle was set at 90°. Superfluous movement of the trunk was controlled via a belt across the abdomen and a crossover shoulder harness. Subjects were positioned with their arms folded across their chest with their thigh securely strapped to the seat. The knee flexion-extension axis was aligned with the dynamometer axis. Both knee extension and flexion strength was measured at 60°, with the reference point being full knee extension (0° = knee fully extended), as adapted from previous studies (Hunter, St Clair Gibson, Lambert et al., 2002; Hunter et al., 2003; Hunter, St Clair Gibson, Mbambo et al., 2002; Kay et al., 2000; Tucker et al., 2004). Calf MVIC was performed on a seated calf-raise machine. Subjects were asked to sit upright in order to keep trunk-thigh angle at 90°; knee flexion-extension angle was set at 90° and ankle planta-dorsi flexion angle was also 90°. For each muscle subjects performed three MVICs. Subjects were verbally encouraged to exert maximal force during each 5-s contraction. Subjects had a 30-s rest between reps and one minute of rest between sets (muscle groups).

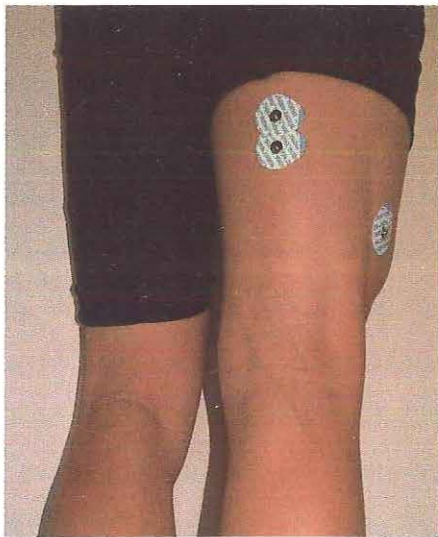
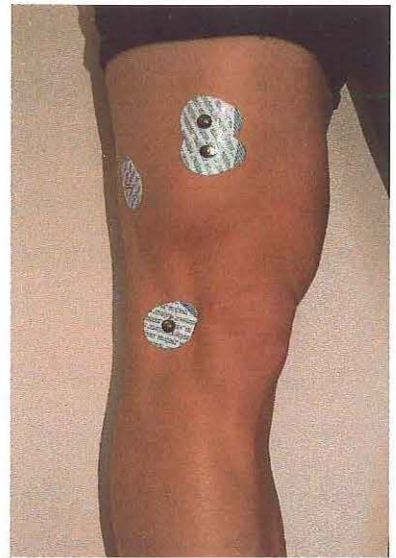
### **3.3.5.2 Muscle activation of the lower limb**

Muscle activation of vastus lateralis, biceps femoris and soleus muscles were recorded via EMG. The MegaWin Muscle Tester ME3000 Professional 8 (Mega Electronics Ltd, Kuopio, Finland) was used to sample EMG at a frequency of 1000 Hz. The data logger was linked to a laptop via a fibre-optic cable and raw EMG was sampled online with the MegaWin software (version 1.2) (Mega Electronics Ltd, Kuopio, Finland). For the MVIC's, EMG was sampled during each of the isometric strength tests for purposes of data normalisation. Two circular (20-mm diameter) Medi Trace™ 200 mini (The Ludlow Company LP; Chicopee, MA, USA) silver/silver chloride (Ag/AgCl), disposable surface electrodes were fitted to the belly of each investigated muscle on the right leg. Inter-electrode distance was 20-mm and all electrodes were positioned and aligned as suggested in the European Recommendations for Surface ElectroMyoGraphy (Hermens et al., 1999).



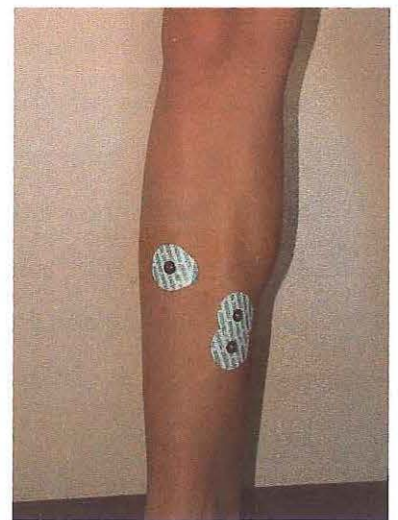
The muscles and their respective electrode placements were as follows:

- Vastus lateralis (VL) -  $\frac{2}{3}$  of the way on the line from the anterior superior iliac spine to the lateral border of the patella. Ground - Lateral epicondyle



- Biceps femoris (BF) -  $\frac{1}{2}$  way along the line from the ischial tuberosity to the lateral epicondyle of the tibia. Ground - Iliotibial tract

- Soleus (Sol.) -  $\frac{2}{3}$  of the way on the line from the medial condyle of the femur to the medial malleolus. Ground -  $\frac{1}{2}$  the way along the line tibial tuberosity to the talus on the anterior margin of the tibia.

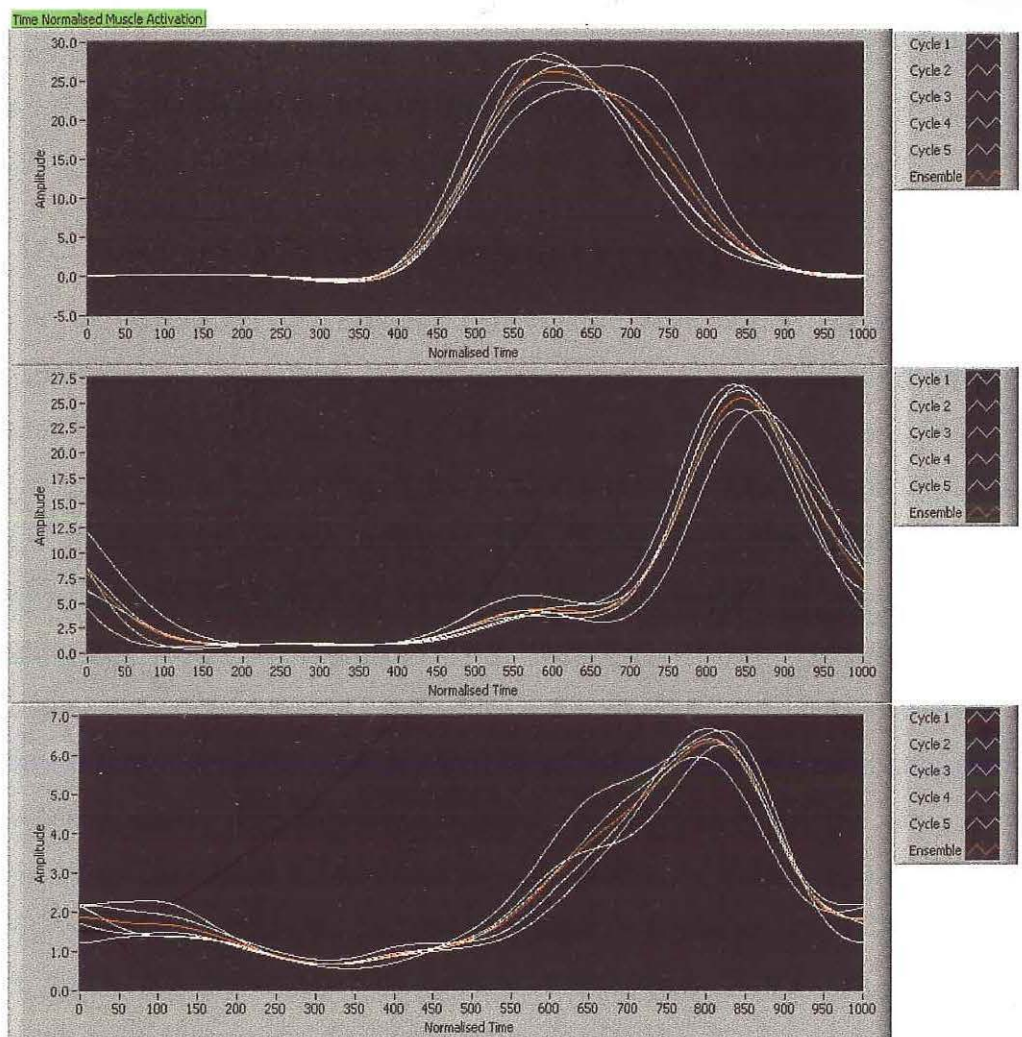


**Figure 11.** Electrode placement of vastus lateralis, biceps femoris and soleus during the 100-km time trials (Hermens et al., 1999).



The skin was prepared prior to electrode placement by shaving the area, then light abrasion, followed by wiping with an alcohol swab to remove oil and dirt. Following skin preparation and electrode placement, skin impedance was determined with less than 5 k $\Omega$  being regarded as acceptable. To minimise movement artefact, Beirsdorf Fixomull™ was placed over the electrodes and the wires. To ensure that the subject's normal cycling pattern was not interrupted, the pre-amplified wires from the EMG were taped to the body and bike using Fixomull™, to avoid any extraneous movement.

A digital switch securely fitted to the bicycle frame was used to identify bottom dead centre (BDC) for the purposes of EMG data time normalisation. The switch allowed for the production of a digital signal ( $\pm$  10Volts) whenever the crank arm reached BDC. EMG data was collected for six seconds mid-way through each of the 1-km sprints (10.5, 32.5, 52.5, 72.5 and 99.5-km), 4-km sprints (22, 42, 62, and 82-km) and at 11 non-sprint distances (1, 5, 15, 28, 26.5, 48, 56.5, 68, 76.5, 88, and 95-km) (St Clair Gibson, Schabort et al., 2001) (Appendix J). Subjects were blinded as to the time that EMG was sampled, and EMG was only sampled while subjects were in a seated position. EMG data from five continuous crank revolutions was used to calculate integrated EMG (iEMG). With the use of LabVIEW graphical development software (version 6.1; National Instruments Corporation, Austin, TX) raw EMG data was full-wave rectified, and passed through a high-pass fourth order Butterworth filter (cut-off frequency of 15 Hz) to remove movement artefact. EMG data was then smoothed with a low-pass fourth order Butterworth filter (cut-off frequency of 5 Hz) to produce a linear envelope (Lepers et al., 2002; Tucker et al., 2004). An ensemble average was generated from the five crank revolutions was then taken from time normalised data (0-1000 points for BDC to BDC) to reduce within subject variability (Figure 12). EMG data were amplitude normalised using the MVIC's. The MVIC value was determined as the greatest value for an averaged 200-ms window of the linear envelope. The greatest EMG value for any of the three MVIC trials was used for normalisation purposes. An iEMG value at each data point was taken as the average of all time-series values in the ensemble average.



**Figure 12.** Typical muscle activation patterns for vastus lateralis (top), biceps femoris (middle) and soleus (bottom) during the five crank revolutions.

### **3.3.6 Cognitive variables**

#### **3.3.6.1 Perceived scales**

During the 100-km time trials, RPE via the 20-point Borg scale (Borg, 1982), thermal sensation (Young et al., 1987), and perceived pain intensity in the quadriceps (Ciubotariu et al., 2004) were recorded at 19 different distances (0, 5, 10.5, 15, 22, 28, 32.5, 36.5, 42, 48, 52.5, 56.5, 62, 68, 72.5, 76.5, 82, 88, 95, and 100-km).

#### **3.3.6.2 IT/RT and multidimensional fatigue inventory tests**

Prior to performing the exercise performance tests during sessions 2-5, subjects performed an inspection (visual process) time (IT) test followed by a reaction time test (RT). In relation to the IT tests, subjects sat on a chair facing a computer monitor. Stimuli consisting of two vertical lines were then presented on the monitor for altering time periods, followed by a “flash” mask. The subjects then indicated whether the lines were of similar or differing lengths by pressing the corresponding key on the keyboard (Burns & Nettelbeck, 2003). Using the same computer, subjects performed the RT test which again involved a stimulus consisting of two vertical lines. However, this stimulus remained on the screen until subjects pressed a corresponding key indicating whether the lines were of the same or differing lengths. This method has been used in previously published studies to assess IT/RT and has found to be a valid and reliable indicator of IT/RT (Burns & Nettelbeck, 2003; Nettelbeck, 2001). Immediately following the IT/RT tests, subjects performed the multidimensional fatigue inventory (Appendix K). Upon completion of all exercise trials, subjects were encouraged to continue cycling at a self-selected power output for a further five minutes. Seven minutes following the trial, subjects repeated the IT/RT test.

### **3.3.7 Fluid intake and weight loss**

During the time-trials, subjects ingested a 6 g/100mL polymer solution at a rate of 750 mL every 45 min, so as to prevent hypoglycemia and dehydration. Solutions containing 8% carbohydrate or less do not inhibit gastric emptying rates (Coyle & Montain, 1992; Dennis et al., 1997). Furthermore, carbohydrate consumption rates in excess of 50-60 g.h<sup>-1</sup> have been shown to be sufficient to

maintain homeostatic blood glucose levels (Coyle & Montain, 1992; Dennis et al., 1997). The solution was stored at 7.6°C prior to being given to the subject at 45 min intervals. Water was consumed *ad libitum* during the time-trials and fluid intake was recorded. Prior to beginning the trial, subjects voided their bladder and body mass was measured with Mettler Toltdo ID1 scales (Columbus, OH, USA). Following completion of the time trial, body mass was again recorded after the wiping of excess sweat with a towel. After compensating for fluid intake, body mass loss during the trial was used to estimate sweat rate. Subjects were instructed to empty their bladder again and were reweighed; post time-trial mass minus post-urination mass was considered to be a measure of urine production rate.

### **3.4 Statistical Analysis**

Each dependant variable throughout the trials (normalised power output, normalised iEMG, temperatures, blood variables, perceived scales, and cardio-respiratory measures) were analysed using a two-way ANOVA with two within-subject variables (condition and distance). Where a significant effect was found between conditions, post-hoc comparisons were made using Turkey's "honesty significant difference" HSD test for pairwise comparisons. All post-hoc tests were performed based on comparing the first data point with proceeding data points. Where a significant difference was found within a condition, the main effect was analysed using the "least significant difference" LSD for pairwise comparisons. Interactions were not examined in this study.

Performance times, average power outputs, multidimensional fatigue inventory scores (MFI-20), pre and post body weights, fluid ingestion, and IT/ RT test scores were analysed using a paired sample *t*-test. Data for both the hot and cold trials were pooled and a Pearson product moment correlation coefficient was calculated to determine relationships between variables. Power and iEMG data are expressed as normalised data, as described above. All statistical tests were conducted using SPSS version 10.0 (Chicago, IL, USA), and data are presented as means and standard deviations. For all analyses, significance was accepted at  $P < 0.05$ .

## CHAPTER 4 RESULTS

Although the various models of fatigue may be interrelated (see Section 2, Figure 9), the results of present study have been presented in relation to the specific physiological, biochemical, biomechanical, and cognitive variables described in Section 1.2.

### 4.1 Subject Characteristics

Table 2 shows the characteristics of the nine subjects participating in the study.

**Table 2.**

Subject characteristics including the mean values and ranges for age, mass, height,  $\dot{V}O_{2\max}$ , and peak power output

	Mean $\pm$ SD	Range
Age, y	31 $\pm$ 6	22 - 41
Mass, kg	75.7 $\pm$ 11.8	54.7 - 90.0
Height, cm	177 $\pm$ 4	170-183
$\dot{V}O_{2\max}$		
mL $\cdot$ kg $^{-1}\cdot$ min $^{-1}$	62.1 $\pm$ 8.5	55.3 - 78.0
L $\cdot$ min $^{-1}$	4.56 $\pm$ 0.74	3.71 - 5.02
Peak power output		
W	335 $\pm$ 23	310 - 363
W $\cdot$ kg $^{-1}$	4.6 $\pm$ 0.7	3.5 - 5.9

## 4.2 Performance Times and Power Output

The time taken to complete the 100-km time trial was significantly greater in the hot trial compared to the cold trial ( $P<0.001$ ; Table 3). The average power output throughout the entire trial was  $258 \pm 27$  W in the cold trial compared to  $219 \pm 41$  W in the hot trial ( $P<0.001$ ). Average power output was also significantly lower in the hot trial compared to the cold trial during the non sprint ( $P<0.001$ ), 1-km sprint ( $P<0.01$ ) and 4-km sprint ( $P<0.001$ ) stages (Table 3).

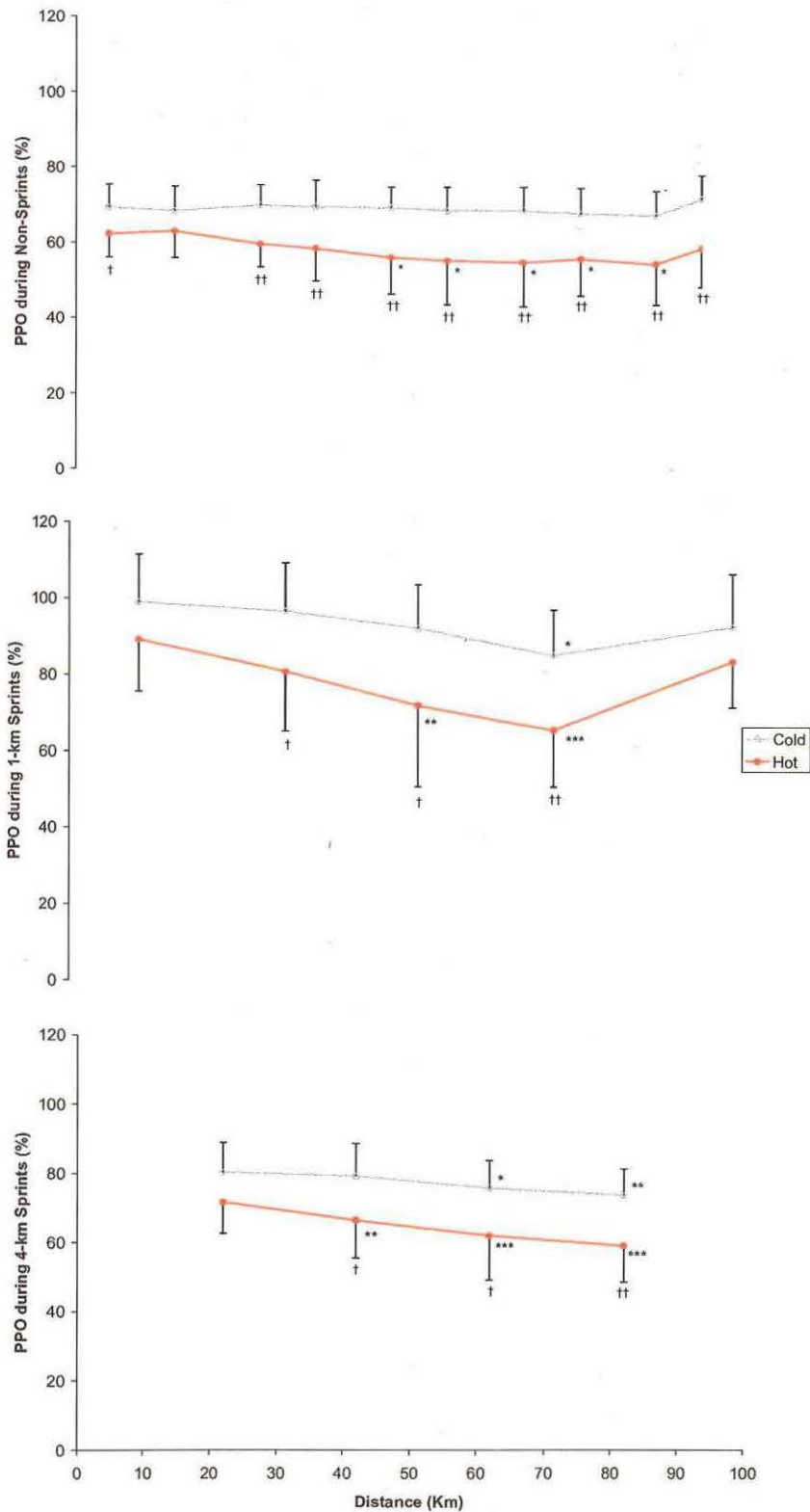
**Table 3.**

Time and average power output during 100-km time trials in cold and hot trials

	Cold	Hot
Time, min	$169 \pm 7$	$181 \pm 13^{**}$
Average power output, W	$258 \pm 27$	$219 \pm 41^{**}$
Non-sprints, W	$232 \pm 26$	$195 \pm 37^{**}$
1-km sprints, W	$313 \pm 32$	$268 \pm 45^*$
4-km sprints, W	$261 \pm 34$	$219 \pm 41^{**}$

Notes: \* denotes significance at  $P<0.01$  vs. cold trial; \*\*denotes significance at  $P<0.001$  vs. cold trial.

Figure 13 shows the normalised power output during the hot and cold time trials averaged throughout the non-sprint, 1-km and 4-km sprint stages. Power output was significantly lower in the hot trial compared to the cold trial during the non-sprint ( $P<0.01$ ), the 1-km ( $P<0.01$ ), and the 4-km ( $P<0.01$ ) sprint stages. During the cold trial, non-sprint power output remained statistically constant throughout the entire trial. Power output was significantly lower during the 1-km sprints at 72-km compared to the first 1-km sprint, while power output during the 4-km sprints was significantly lower at 62-km and 82-km compared to the first 4-km sprint (Figure 13). In the hot trial, non-sprint power output was significantly reduced compared to the first non-sprint time point from 48-km and beyond. During the hot trial, power output during the 1-km and 4-km sprints was also significantly decreased from 52.5-km and 42-km, respectively, and beyond, with the exception that power output during the final 1-km sprint (99-km) was similar to that of the first 1-km sprint (10-km) in both the hot and cold trial. Power output during the final 1-km sprint was also not significantly different between the hot and cold trials.



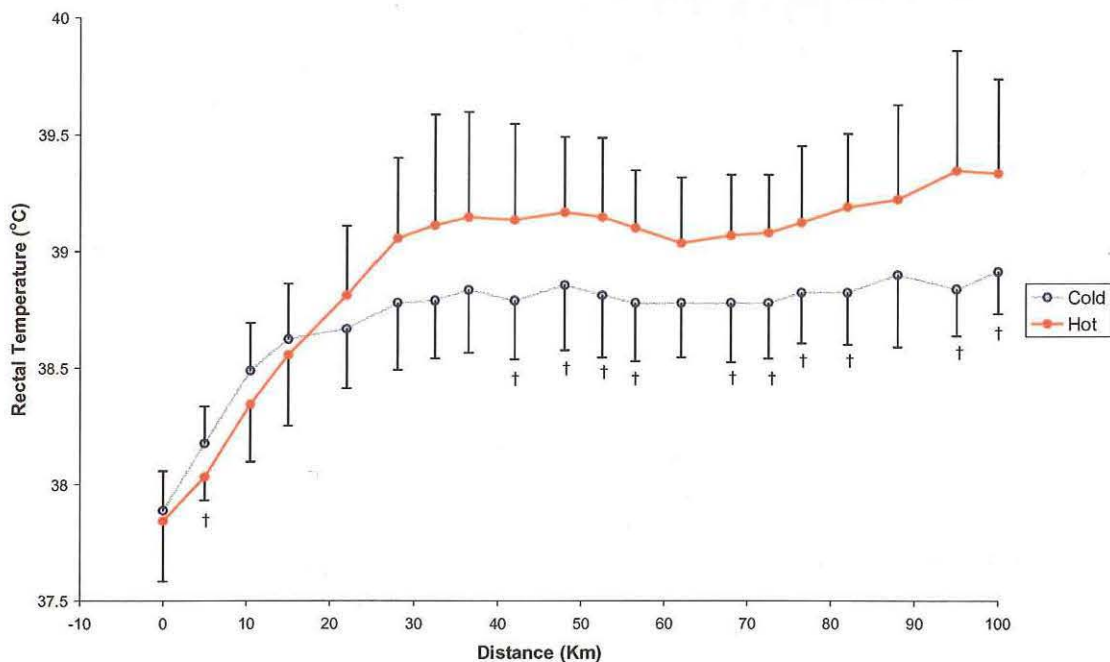
**Figure 13.** Percentage of peak power output (PPO) during the non-sprint, 1-km and 4-km sprint phases in both the hot and cold trials. \* $P < 0.05$ , distance main effect for the same environmental condition; \*\* $P < 0.01$ , distance main effect for the same environmental condition; \*\*\* $P < 0.001$ , distance main effect for the same environmental condition. † $P < 0.05$ , vs. hot trial; †† $P < 0.01$ , vs. hot trial.



### 4.3 Physiological Variables

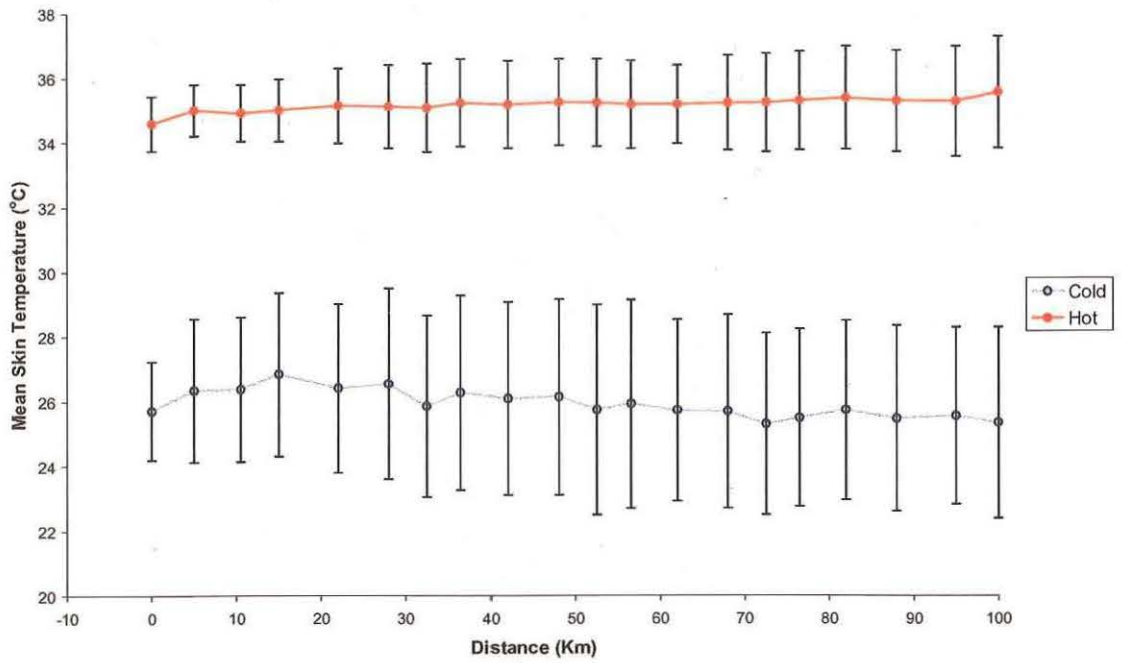
#### 4.3.1 Thermoregulatory responses

Rectal temperature increased significantly over distance ( $P<0.001$ ) in both the hot and cold trials (Figure 14a). Interestingly, rectal temperature initially increased at a greater rate in the cold trial and was significantly higher compared to that of the hot trial at 5-km ( $P<0.05$ ). Rectal temperature increased significantly over distance in the hot trial and was significantly greater compared to the cold trial at 28-km and beyond ( $P<0.05$ ), with the exception of 62-km ( $P=0.053$ ) and 88-km ( $P=0.077$ ). Figure 14b shows the mean skin temperature in both hot and cold trials. Mean skin temperature increased significantly over distance ( $P<0.001$ ) in both the hot and cold trials and was significantly higher in the hot compared to the cold condition at all distances ( $P<0.001$ ). Mean body temperature increased significantly over distance ( $P<0.001$ ) in both the hot and cold trials and was significantly higher in the hot compared to the cold condition at all distances (all  $P<0.001$ ; Figure 14c).

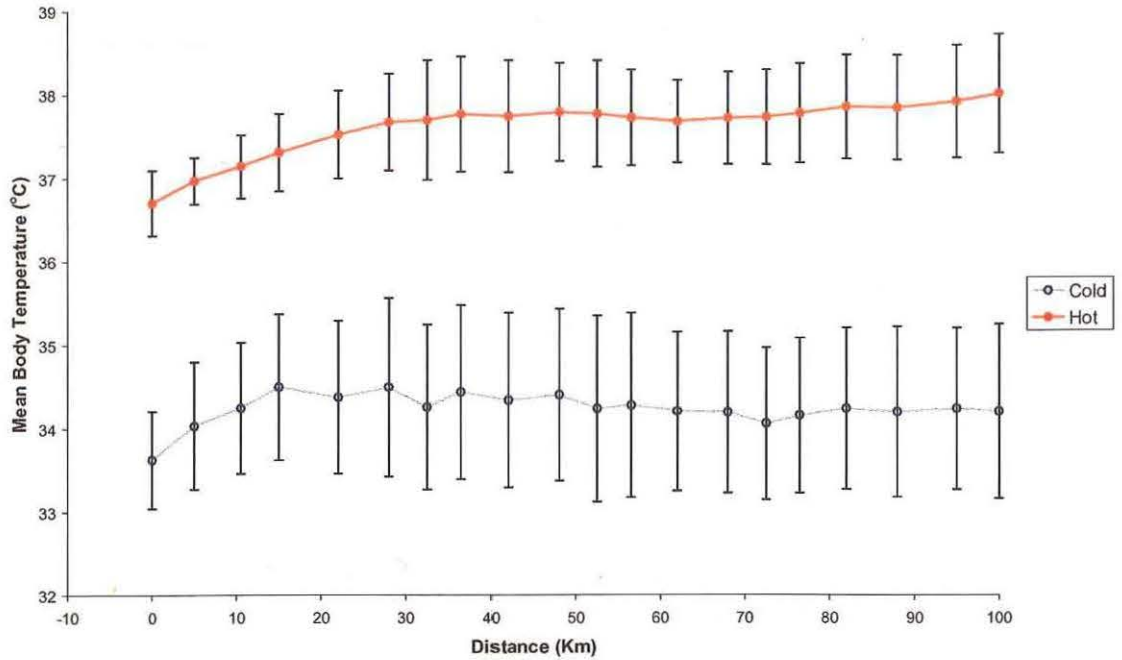


**Figure 14a.** Rectal temperature during the 100-km time trial in both hot and cold conditions. † $P<0.05$  vs. hot trial.





**Figure 14b.** Mean skin temperature during the 100-km time trial in both hot and cold conditions. Mean skin temperature was significantly higher in the hot versus the cold trial at all distances.

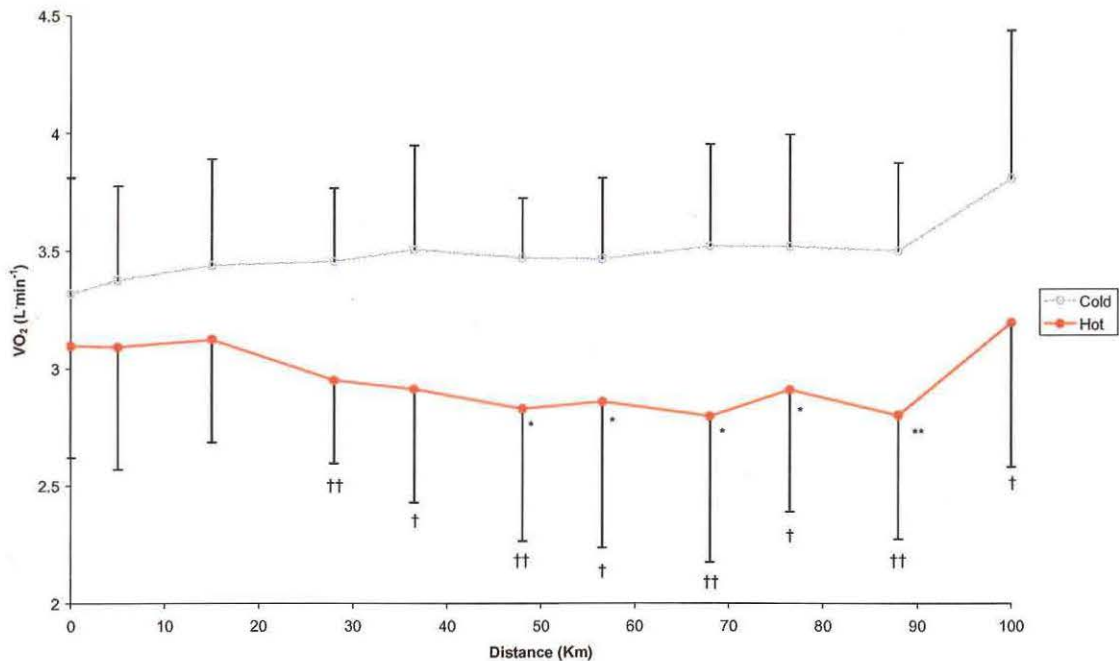


**Figure 14c.** Mean body temperature during the 100-km time trial in both hot and cold conditions. Mean body temperature increased significantly over distance in both the hot and cold trials ( $P < 0.001$ ). Mean body temperature was significantly higher in the hot versus the cold trial at all distances.

### 4.3.2 Cardiorespiratory measures

#### 4.3.2.1 Oxygen consumption

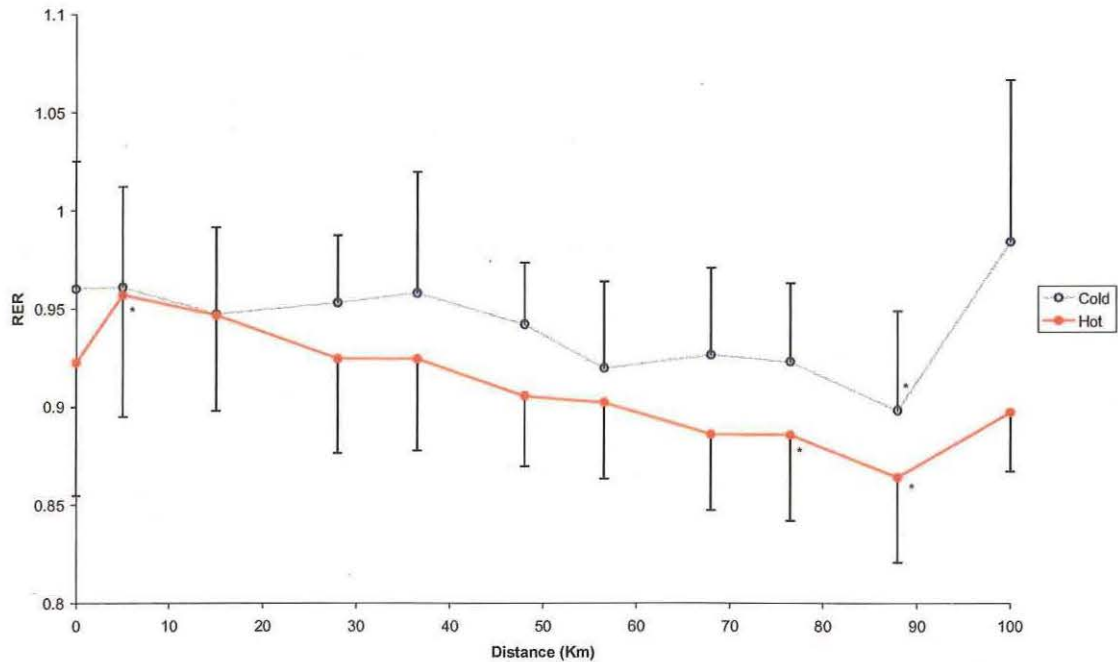
$\dot{V}O_2$  was significantly lower in the hot compared to the cold trial at 28, 48, 68, 88, and 100-km ( $P<0.05$ ).  $\dot{V}O_2$  significantly declined with increasing distance compared to starting  $\dot{V}O_2$  in the hot trial at 48, 58, 68, 78, and 88-km ( $P<0.05$ ), but did not change throughout the 100-km cold trial (Figure 15). The mean fractional utilisation of  $\dot{V}O_{2\max}$  during the non-sprint stages of the 100-km time trials was  $64 \pm 10\%$  and  $76 \pm 9\%$  in the hot and cold trials, respectively. The peak  $\dot{V}O_2$  obtained during the final 1-km sprint and was  $70 \pm 11\%$  and  $83 \pm 12\%$  of  $\dot{V}O_{2\max}$  in the hot and cold trials, respectively.



**Figure 15.** Oxygen uptake ( $\dot{V}O_2$ ) during a 100-km time trial in hot versus cold conditions. \* $P<0.05$ , distance main effect for the same environmental condition; \*\* $P<0.01$ , distance main effect for the same environmental condition. † $P<0.05$ , vs. cold trial; †† $P<0.01$ , vs. cold trial.

#### 4.3.2.2 Respiratory exchange ratio (RER)

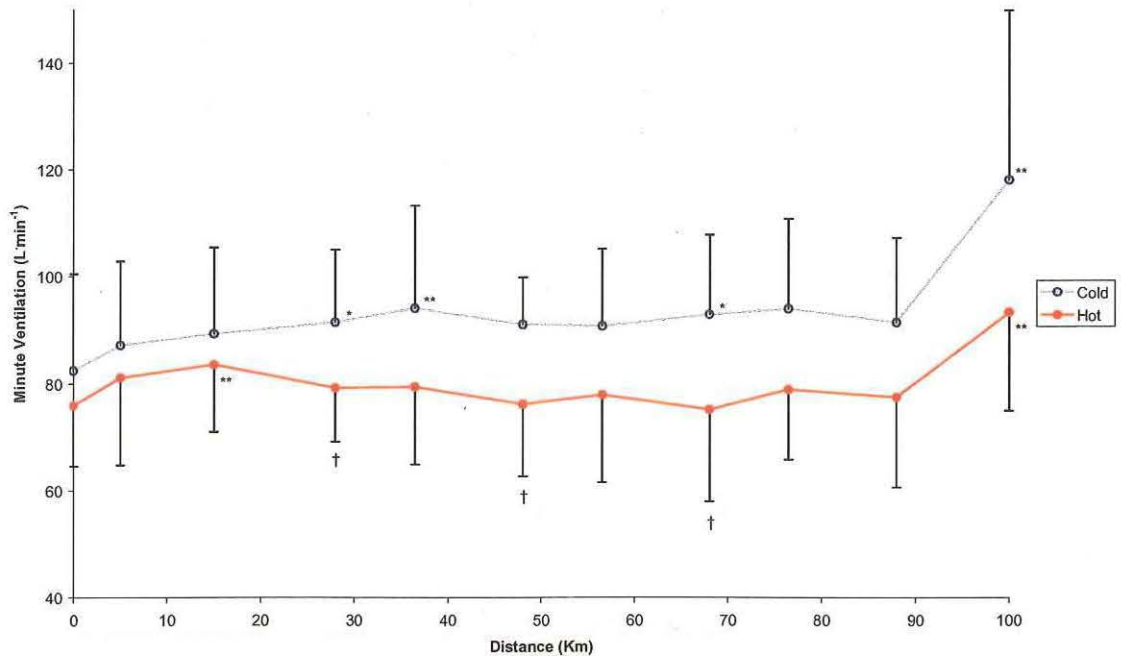
Respiratory exchange ratio (RER) was not significantly different between hot and cold trials. RER was significantly lower at 78 and 88-km compared to RER values at the start of the trial (0-km) in the hot and cold trials, respectively. Non-sprint RER values averaged  $0.94 \pm 0.05$  in the cold and  $0.91 \pm 0.05$  in the hot. RER values ranged from  $0.90 \pm 0.05$  to  $0.98 \pm 0.08$  in the cold and  $0.89 \pm 0.04$  to  $0.96 \pm 0.06$  in the hot.



**Figure 16.** Respiratory exchange ratio (RER) during a 100-km time trial in both hot and cold conditions. \* $P < 0.05$ , distance main effect for the same environmental condition. There was no significant difference for RER between hot and cold trials.

#### 4.3.2.3 Minute ventilation

$\dot{V}_E$  was significantly lower in the hot trial at 28, 48 and 68-km ( $P < 0.01$ ). In the cold trial,  $\dot{V}_E$  was significantly increased at 28, 36.5 and 68-km compared to the start of the trial (0 km).

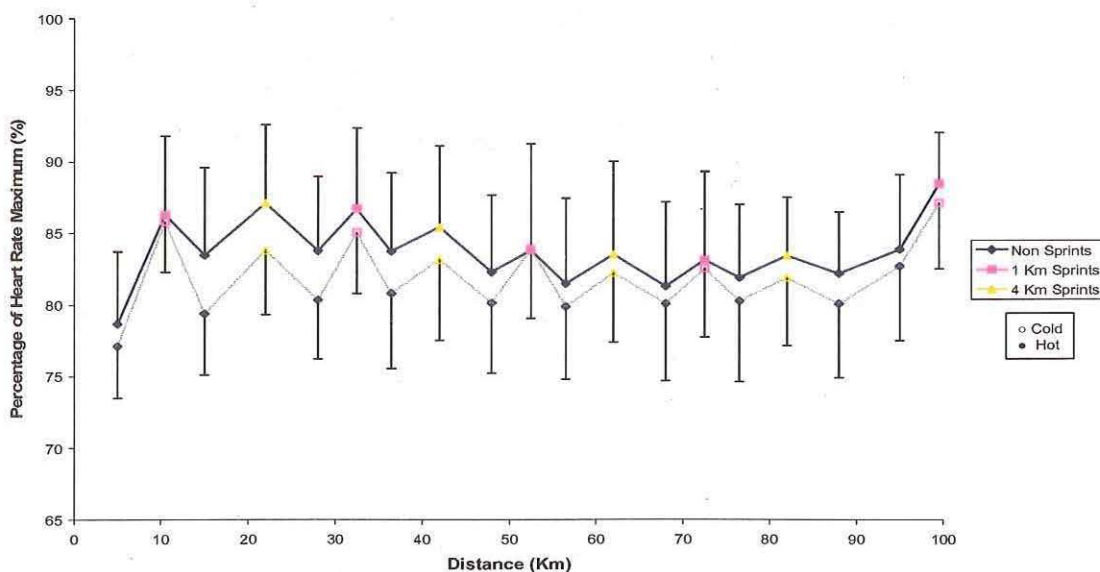


**Figure 17.** Minute ventilation ( $\dot{V}_E$ ) during a 100-km time trial in hot and cold conditions. \* $P < 0.05$ , distance main effect for the same environmental condition. † $P < 0.05$ , vs. cold trial.

#### 4.3.2.4 Heart rate

Figure 18 shows the heart rate averaged over the non-sprints, 1-km and 4-km sprints. Heart rate increased significantly in both the hot ( $P < 0.001$ ) and cold ( $P < 0.001$ ) trials compared to that at 5-km. Heart rate was not significantly different in the hot compared to the cold trial. The average peak heart rate reached during the final 1-km sprint and was  $89 \pm 4\%$  ( $170 \pm 8$  bpm) and  $87 \pm 5\%$  ( $167 \pm 9$  bpm) of its maximum in the hot and cold trials, respectively.

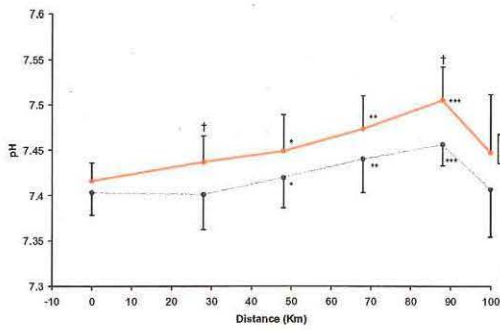




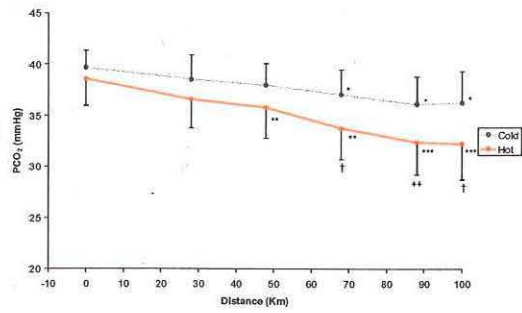
**Figure 18.** Fractional utilisation of maximum heart rate during a 100-km time trial in both hot and cold conditions.

#### 4.4 Biochemical blood variables

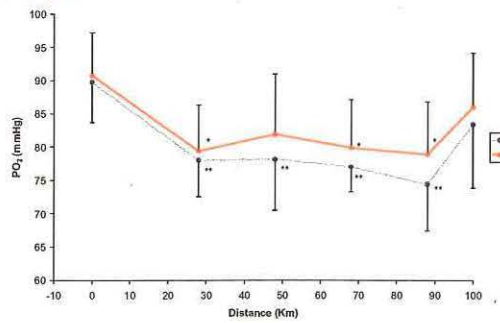
Figure 18a shows that pH gradually increased over distance in both the hot and cold trials and was significantly higher than post warm-up values at 48-km and beyond in both trials ( $P < 0.05$ ). pH statistically returned to post warm-up values, however, after the final sprint in both hot and cold conditions. pH was lower in the cold trial at 28-km and 88-km compared to the hot trial ( $P < 0.05$ ).  $PCO_2$  fell with increasing distance in both trials and was significantly lower in the hot compared to the cold trial at 68-km and 88-km. In relation to post warm-up values,  $PCO_2$  was significantly lower at 48-km ( $P < 0.01$ ) and 68-km ( $P < 0.05$ ) in both the hot and cold conditions, respectively (Figure 19b). Sodium was significantly lower in the hot compared to the cold trial at all time points, except post warm-up (Figure 19h). From Figure 19i it can be seen that potassium increased over distance in both the hot and cold trials ( $P < 0.01$ ). Urea also increased significantly over distance in both the hot ( $P < 0.05$ ) and cold ( $P < 0.01$ ) trials. Chloride, bicarbonate, glucose,  $sO_2$  and hematocrit remained statistically stable throughout the 100-km time trials. Noticeably, many other blood variables (i.e., pH,  $PO_2$ ,  $HCO_3^-$  and lactate) significantly changed directly following the 100-km final sprint (Figure 19). Lactate was not significantly different both within and between trials until the final sprint when lactate significantly increased in both the hot ( $P < 0.001$ ) and the cold ( $P < 0.01$ ) trials.



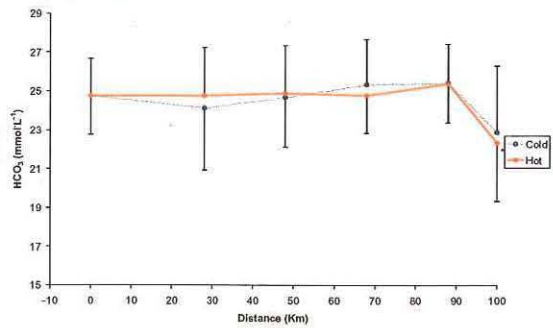
(a) pH



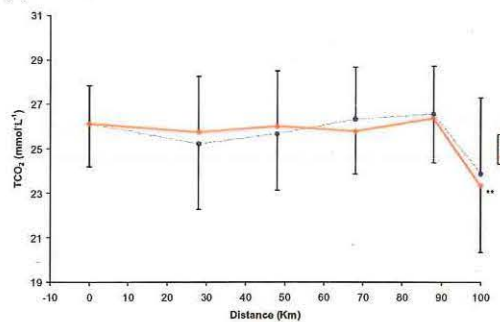
(b) PCO<sub>2</sub>



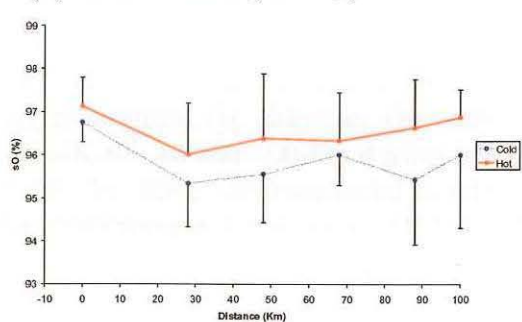
(c) PO<sub>2</sub>



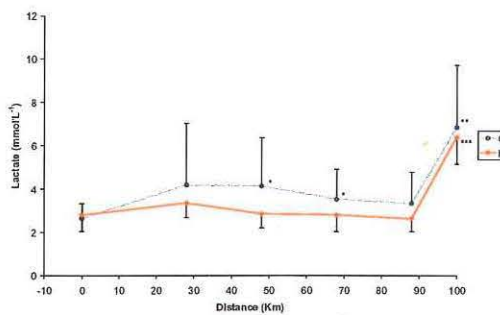
(d) Bicarbonate (HCO<sub>3</sub><sup>-</sup>)



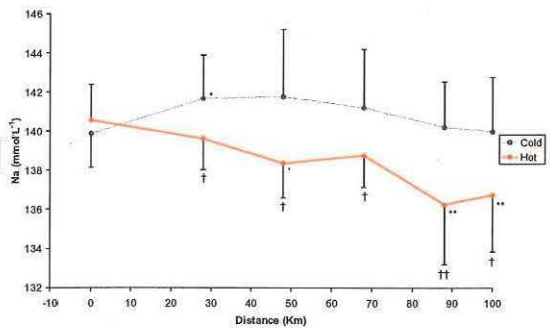
(e) Total carbon dioxide (TCO<sub>2</sub>)



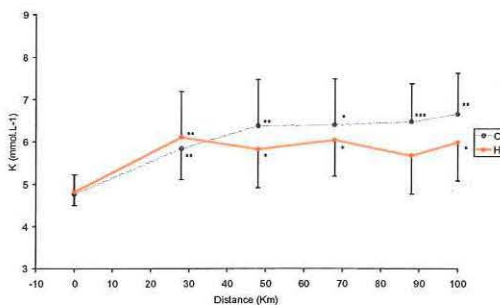
(f) Oxygen saturation (sO<sub>2</sub>)



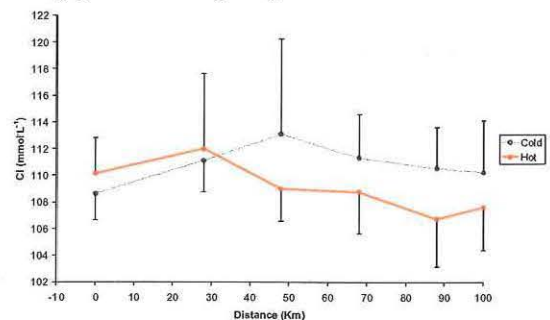
(g) Lactate



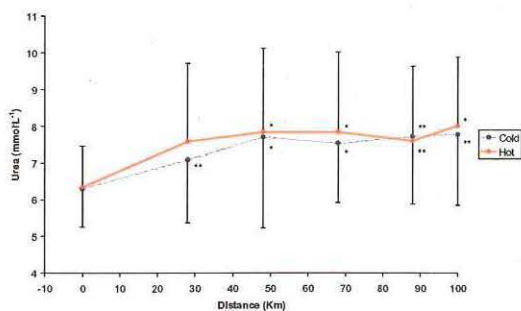
(h) Sodium (Na<sup>+</sup>)



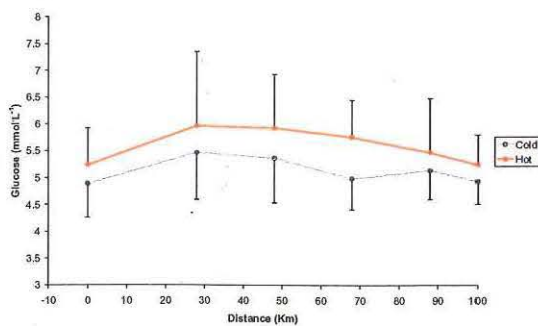
(i) Potassium (K<sup>+</sup>)



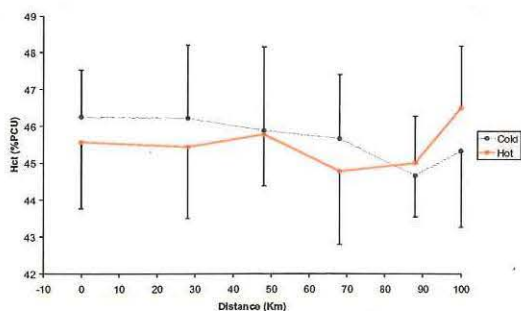
(j) Chlorine (Cl<sup>-</sup>)



(k) Urea



(l) Glucose



(m) Hematocrit (Hct)

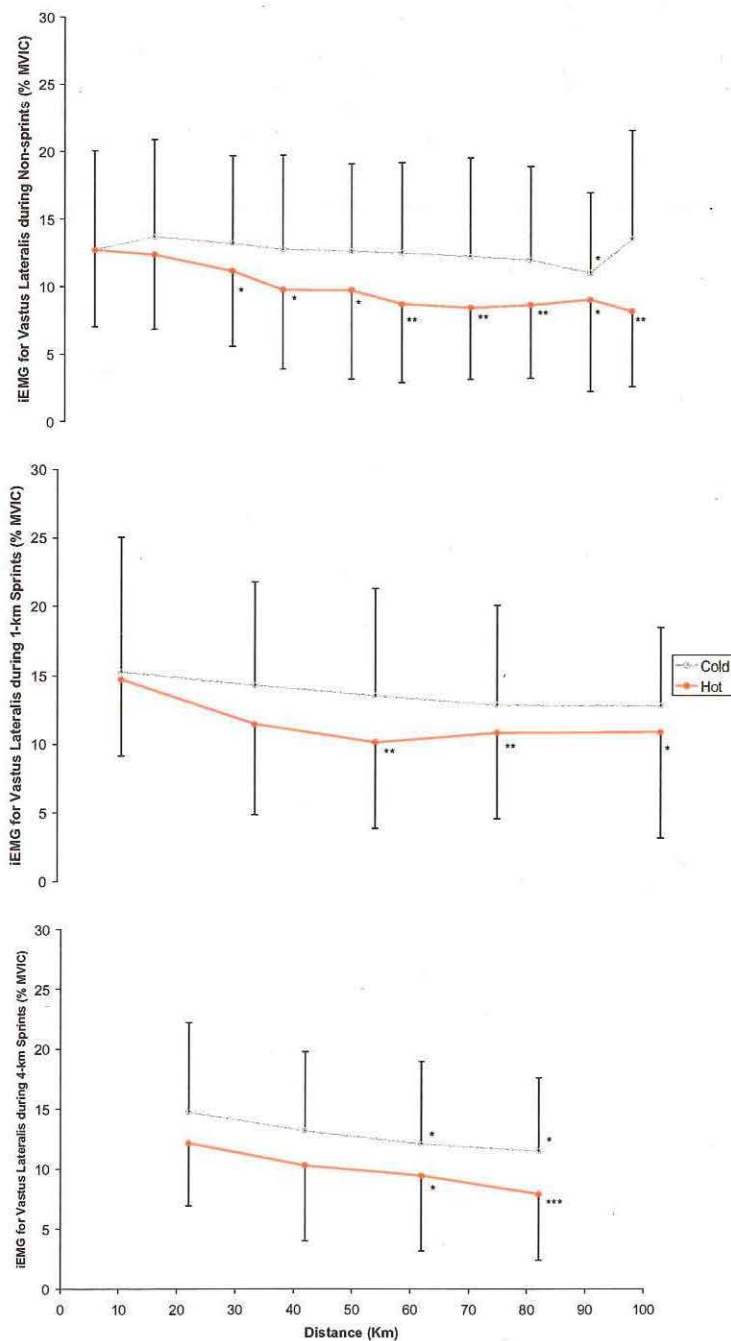
**Figure 19.** Blood biochemical markers (a) pH, (b)  $\text{PCO}_2$ , (c)  $\text{PO}_2$ , (d)  $\text{HCO}_3^-$ , (e)  $\text{TCO}_2$ , (f)  $\text{sO}_2$ , (g) lactate, (h) sodium, (i) potassium, (j) chlorine, (k) urea, (l) glucose, and (m) Hct at post warm-up (0), 28, 48, 68, 88 and 100-km during hot and cold trials. \* $P < 0.05$ , distance main effect for the same environmental condition; \*\* $P < 0.01$ , distance main effect for the same environmental condition; \*\*\* $P < 0.001$ , distance main effect for the same environmental condition. † $P < 0.05$ , vs. hot trial; †† $P < 0.01$ , vs. hot trial; ††† $P < 0.001$ , vs. hot trial.

## **4.5 Biomechanical variables**

### **4.5.1 iEMG amplitude of vastus lateralis**

Figure 20 shows the iEMG amplitude of vastus lateralis during the non-sprint, 1-km and 4-km sprint stages as a percentage of peak iEMG obtained during MVICs. iEMG was not significantly different between the two conditions during either non-sprint, 1-km or 4-km sprints. In the cold trial, non-sprint iEMG was significantly decreased at 88-km ( $P<0.05$ ), whereas non-sprint iEMG decreased from 28-km and beyond in the hot trial ( $P<0.05$ ). During the 1-km sprints, iEMG of vastus lateralis did not change significantly in the cold trial, but gradually decreased in the hot trial and was significantly lower from 52.5-km and beyond ( $P<0.01$ ). During both the hot and cold trials, iEMG of vastus lateralis was significantly different compared to that of the first 4-km sprint from 62-km and beyond ( $P<0.05$ ). iEMG of vastus lateralis ranged from a peak activation of  $15.3 \pm 9.8\%$  MVIC to a minimum activation of  $7.9 \pm 5.5\%$  MVIC in both trials.

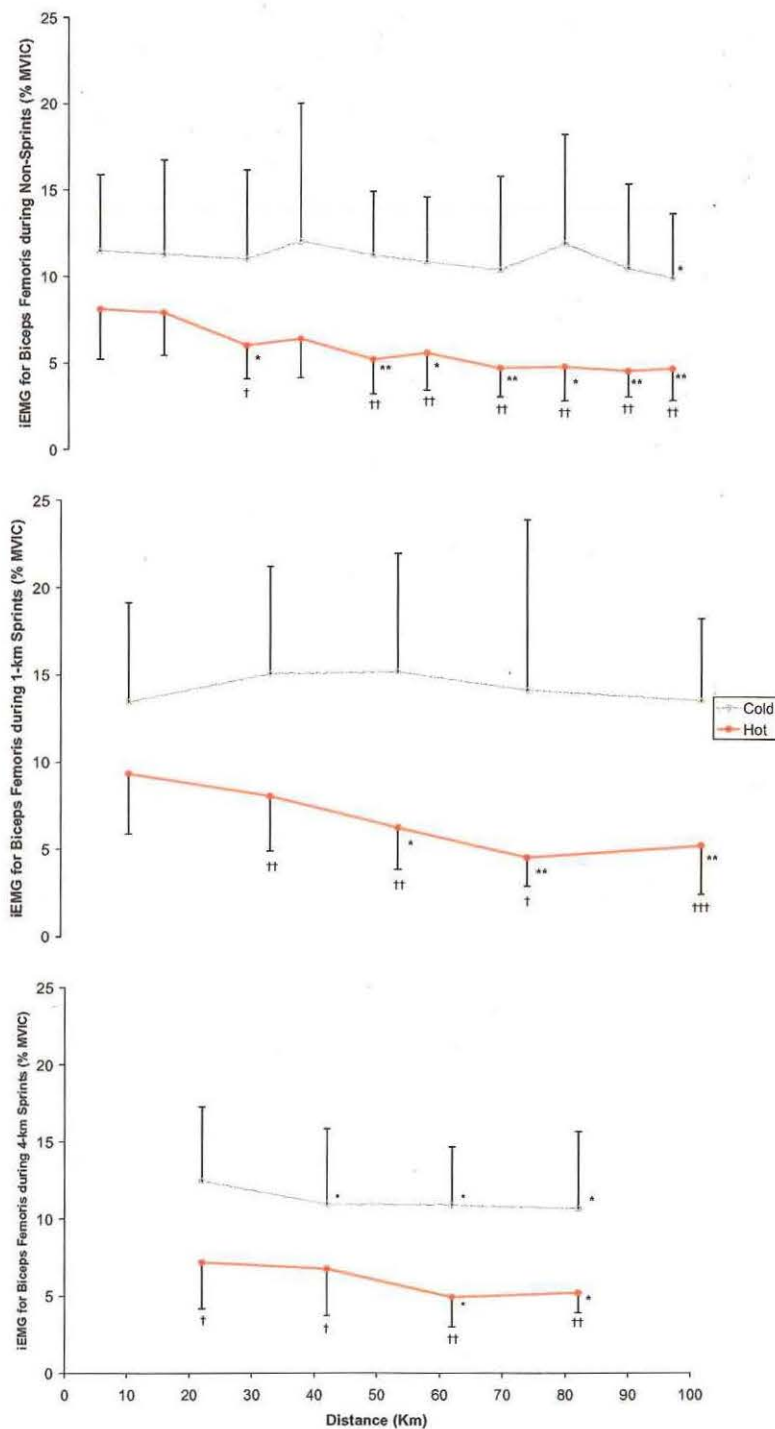




**Figure 20.** Integrated electromyography (iEMG) of vastus lateralis during the non-sprint, 1-km and 4-km sprints stages in both the hot and cold trials. \* $P < 0.05$ , distance main effect for the same environmental condition; \*\* $P < 0.01$ , distance main effect for the same environmental condition; \*\*\* $P < 0.001$ , distance main effect for the same environmental condition. iEMG was not significantly different between the hot and cold trials.

#### **4.5.2 iEMG amplitude of biceps femoris**

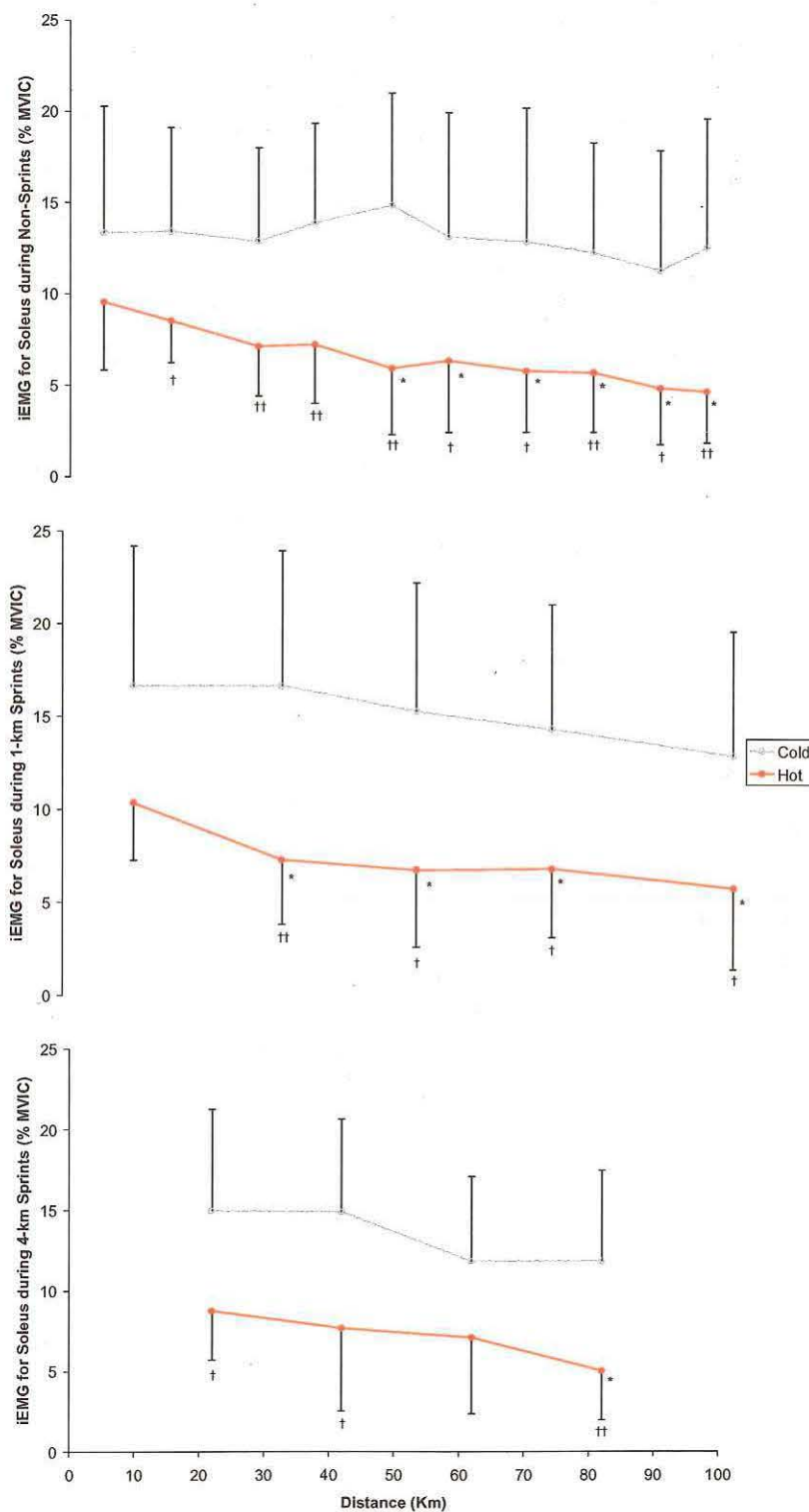
iEMG of biceps femoris was significantly reduced in the hot trial compared to the cold trial during the non-sprints ( $P<0.01$ ), 1-km sprints ( $P<0.01$ ), and during the 4-km sprints ( $P<0.01$ ) (Figure 15). During the cold trial, iEMG of biceps femoris decreased during the 4-km sprints and during the non-sprints and was significantly lower at 42-km and 95-km, respectively (both  $P<0.05$ ). In the hot trial, iEMG of biceps femoris gradually decreased over distance and was significantly different at 28-km, 52.5-km and 62-km during the non-sprints, 1-km sprints and the 4-km sprints, respectively (all  $P<0.05$ ). iEMG of biceps femoris ranged from a peak activation of  $15.2 \pm 6.8\%$  MVIC to a minimum activation of  $4.5 \pm 1.5\%$  MVIC in both trials.



**Figure 21.** Integrated electromyography (iEMG) of biceps femoris during the non-sprint, 1-km and 4-km sprint stages in both the hot and cold trials. \* $P < 0.05$ , distance main effect for the same environmental condition; \*\* $P < 0.01$ , distance main effect for the same environmental condition; \*\*\* $P < 0.001$ , distance main effect for the same environmental condition. † $P < 0.05$ , vs. hot trial; †† $P < 0.01$ , vs. hot trial; ††† $P < 0.001$ , vs. hot trial.

#### **4.5.3 iEMG amplitude of soleus**

iEMG of soleus was significantly reduced in the hot compared to the cold trial during the non-sprints, 1-km sprints, and during the 4-km sprints (all  $P < 0.01$ ; Figure 16). In the hot trial, iEMG of soleus decreased significantly over distance and was significantly different to preceding intervals from 32.5-km, 48-km, and 82-km and beyond for the 1-km sprints, the non-sprints, and the 4-km sprints, respectively (all  $P < 0.05$ ; Figure 22). iEMG of soleus did not significantly change over distance in the cold trial. iEMG of soleus ranged from a peak activation of  $16.6 \pm 7.5\%$  MVIC to minimum activation of  $4.6 \pm 2.8\%$  MVIC in both of the trials.

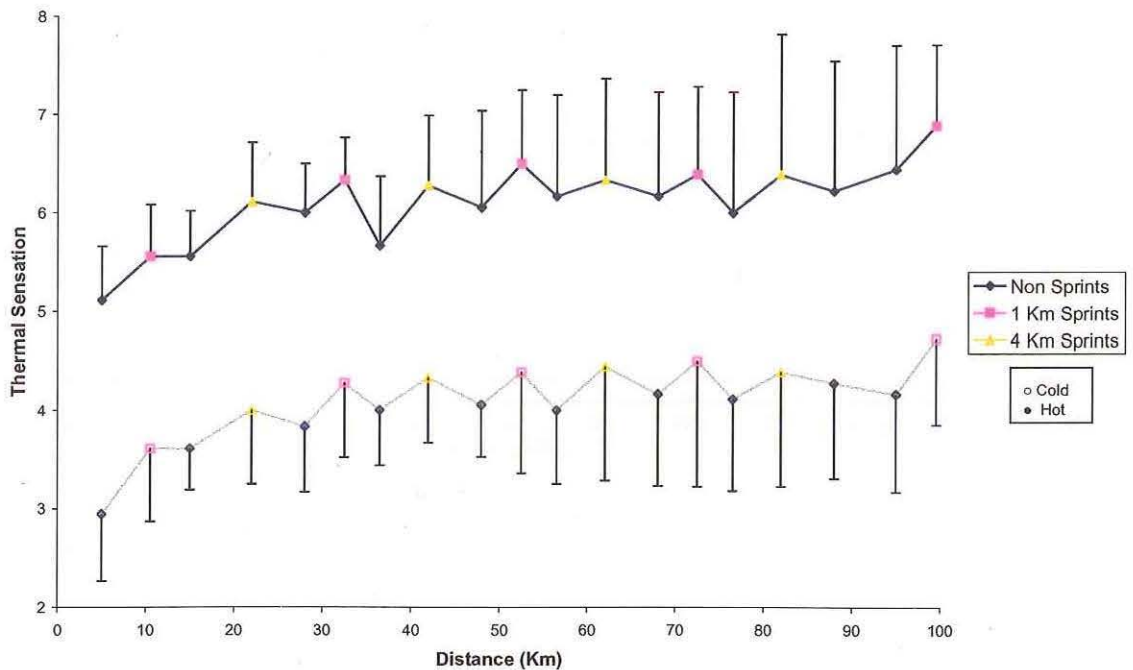


**Figure 22.** Integrated electromyography (iEMG) of soleus during the non-sprint, 1-km sprint stages in both the hot and cold trials. \* $P < 0.05$ , distance main effect for the same environmental condition. † $P < 0.05$ , vs. hot trial; †† $P < 0.01$ , vs. hot trial; ††† $P < 0.001$ , vs. hot trial.

## 4.6 Cognitive Variables

### 4.6.1 Perceived thermal sensation

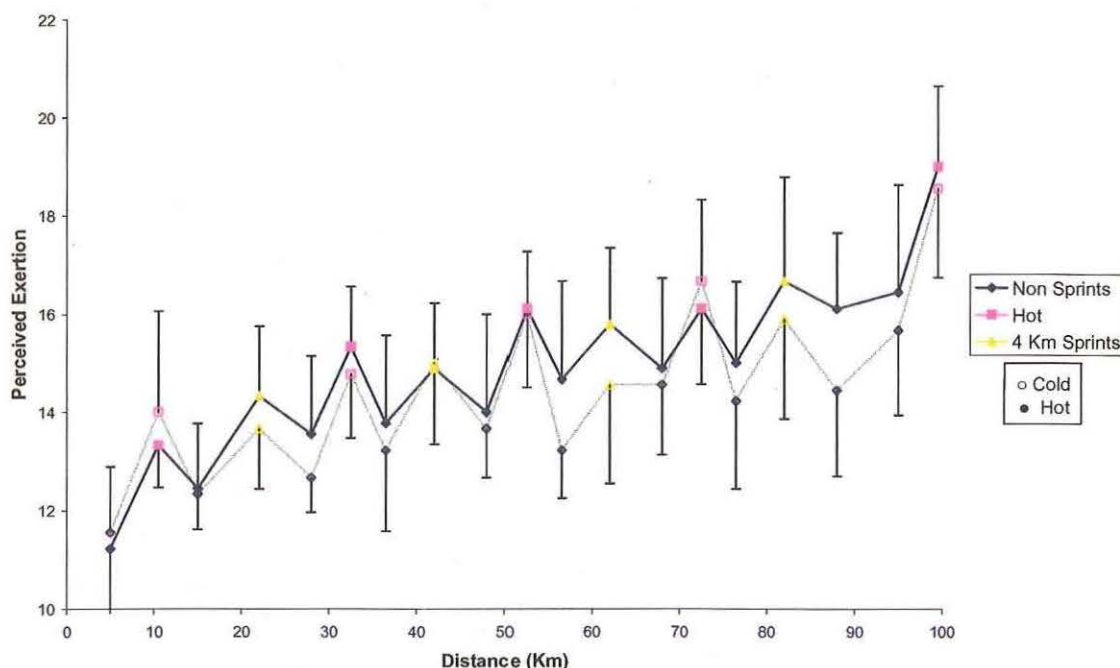
Thermal sensation increased significantly over distance in both the hot ( $P<0.05$ ) and cold ( $P<0.01$ ) trials and was significantly greater in the hot compared to the cold trial ( $P<0.001$ ; Figure 23). Thermal sensation ranged from  $5.1 \pm 0.6$  to  $6.9 \pm 0.8$  and  $2.9 \pm 0.7$  to  $4.7 \pm 0.9$  Thermal Units in the hot and cold trials respectively (see Appendix H for scale).



**Figure 23.** Perceived thermal sensation during a 100-km time trial in hot and cold conditions. Thermal sensation increased significantly over distance in both the hot ( $P<0.05$ ) and cold ( $P<0.01$ ) trials, and was significantly greater in the hot trial compared to the cold trial ( $P<0.001$ ).

#### 4.6.2 Rating of perceived exertion

Rating of perceived exertion (RPE) increased significantly over distance in both the hot ( $P<0.001$ ) and cold ( $P<0.001$ ) trials, but was not significantly different between the hot and cold trials (Figure 24). RPE during the final sprint in the hot and cold were  $19.0 \pm 1.6$  and  $18.6 \pm 1.8$  Borg Units, respectively.

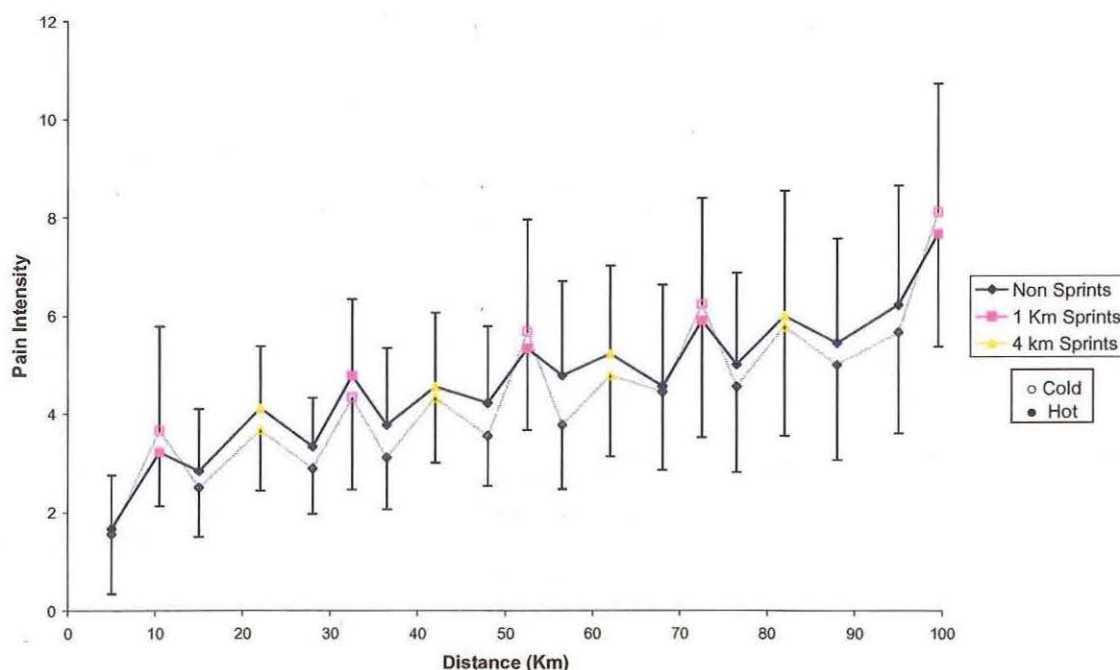


**Figure 24.** Rating of perceived exertion (RPE) during a 100-km time trial in hot and cold conditions. RPE increased significantly over distance in both the hot ( $P<0.001$ ) and cold ( $P<0.001$ ) trials. No significant differences between trials.



### 4.6.3 Perceived pain intensity

Perceived pain intensity in the quadriceps increased significantly over distance in both the hot ( $P<0.001$ ) and cold ( $P<0.001$ ) trials. Pain intensity was not significantly different between the hot and cold trials. During the final sprint, perceived pain intensity was  $7.7 \pm 2.3$  and  $8.1 \pm 2.7$  Pain Units in the hot and cold trials, respectively.



**Figure 25.** Perceived pain intensity of the quadriceps during a 100-km time trial in hot and cold conditions. Perceived pain intensity increased significantly over distance in both the hot ( $P<0.001$ ) and cold ( $P<0.001$ ) trials. No significant differences between trials.



#### 4.6.4 Inspection time and reaction time

Inspection time (IT) and reaction time (RT) were not significantly different following 100-km time trials in either hot or cold conditions compared to pre-trial values (Table 4). Pre- and post-trial IT and RT scores were also not significantly different between the hot and cold trials.

**Table 4.**

Inspection time (IT) and reaction time (RT) measured pre- and post-trial during the hot and cold trials. No significant differences between pre- versus post-trial, or between hot and cold trials.

	Cold		Hot	
	Pre	Post	Pre	Post
IT, ms	2.21 ± 0.51	2.14 ± 0.61	2.28 ± 0.88	2.22 ± 0.94
RT, ms	474 ± 76	482 ± 92	458 ± 63	453 ± 64

#### 4.6.5 Multidimensional fatigue inventory

Table 5 shows the mean score for each component of the multidimensional fatigue inventory (MFI-20) completed prior to the hot and cold trials. Perceived physical fatigue was significantly greater prior to the hot trial than the cold trial ( $P < 0.05$ ). No other component of the inventory was significantly different between the two trials.

**Table 5.**

Scores for the multidimensional fatigue inventory prior to the cold and hot trials

	Cold	Hot
General Fatigue	8.9 ± 3.4	8.6 ± 2.2
Physical Fatigue	6.8 ± 3.0	8.2 ± 3.5*
Reduced Activity	5.7 ± 1.8	6.9 ± 3.9
Reduced Motivation	7.4 ± 2.9	7.1 ± 2.7
Mental Fatigue	8.7 ± 2.3	8.6 ± 3.5

**Notes:** \* denotes significance at  $P < 0.05$  vs. cold trial. Scores can range from a minimum of 4 to a maximum of 20, where a higher score indicates more fatigue.

## 4.7 Fluid Intake and Weight Loss

**Table 6.**

Body mass, fluid intake, weight loss and urine production during 100-km time trial in hot and cold conditions

	Cold	Hot
Pre-trial body mass, kg	76.5 ± 12.2	76.8 ± 11.6
Post-trial body mass, kg	76.6 ± 12.3	76.8 ± 12.0
Fluid intake, L	2.73 ± 0.53	5.14 ± 0.81**
Fluid loss, L	2.78 ± 0.77	5.08 ± 1.33**
Rate of body water loss, L·h <sup>-1</sup>	0.99 ± 0.27	1.68 ± 0.44*
Urine production, L	0.43 ± 0.30	0.28 ± 0.19

Notes: \* denotes significance at  $P < 0.05$  vs. cold trial; \*\* denotes significance at  $P < 0.01$  vs. cold trial. Fluid loss accounts for fluid intake and is calculated based on changes in body mass, sweat loss, and urine production.

Fluid intake, sweat loss, and rate of body water loss were significantly greater in the hot compared to the cold condition (all  $P < 0.05$ ; Table 6). Neither pre-body mass, post-body mass, nor urine production were significantly different between the hot and cold trials.

## 4.8 Correlations between Variables

### 4.8.1 Power output and iEMG

Reductions in power output showed a moderate positive correlation to reductions in iEMG of biceps femoris during the non-sprints, 1-km, and 4-km sprints (all  $P < 0.01$ ; Table 7). Reductions in power output were also weakly correlated to a reduced iEMG of soleus, during all intensities (all  $P < 0.05$ ). No significant correlations were found between iEMG of vastus lateralis and power output during the non-sprints, 1-km, or 4-km sprints (Table 7).

**Table 7.**

Correlation coefficient (r) between reductions in power output and iEMG of vastus lateralis, biceps femoris, and soleus during non-sprints, 1-km and 4-km sprints.

	Power		
	Non-Sprints	1-km Sprints	4-km Sprints
iEMG of vastus lateralis	-0.23	0.09	-0.00
iEMG of biceps femoris	0.52**	0.45**	0.45**
iEMG of soleus	0.23**	0.26*	0.27*

Notes: \* denotes significance at  $P < 0.05$  level; \*\* denotes significance at  $P < 0.01$  level.

#### **4.8.2 iEMG of vastus lateralis, biceps femoris and soleus**

Table 8 shows the relationship between iEMG of vastus lateralis and iEMG of biceps femoris and soleus. iEMG of vastus lateralis was positively correlated to iEMG of biceps femoris during the non-sprints, 1-km and 4-km sprints (all  $P < 0.01$ ; Table 8). A positive correlation was also found to exist between iEMG of vastus lateralis and soleus during the non-sprints, 1-km and 4-km sprints (all  $P < 0.01$ ; Table 8).

**Table 8.**

Correlation coefficient (r) between power output and iEMG of vastus lateralis, biceps femoris, and soleus during non-sprints, 1-km and 4-km sprints.

	EMG	
	Biceps femoris	Soleus
Non-sprints	0.37**	0.39**
1-km sprints	0.33**	0.46**
4-km sprints	0.45**	0.41**

#### 4.8.3 iEMG and biochemical markers in the blood

Table 9 shows the correlations between blood variables pH, lactate, bicarbonate ion ( $\text{HCO}_3^-$ ), sodium ( $\text{Na}^+$ ), and potassium ( $\text{K}^+$ ) and iEMG of vastus lateralis, biceps femoris, and soleus (Table 9). There was a weak positive correlation between iEMG of all muscle examined and pH. Positive correlations were also found between iEMG of biceps femoris, blood sodium ( $r=0.31$ ;  $P<0.01$ ), and also potassium ( $r=0.24$ ;  $P<0.05$ ). iEMG of biceps femoris was also negatively correlated with blood lactate and  $\text{HCO}_3^-$  concentrations. Both blood bicarbonate ion ( $\text{HCO}_3^-$ ) and blood lactate were correlated to pH and ( $r=0.51$ ;  $P<0.01$ ) and ( $r=-0.42$ ;  $P<0.01$ ), respectively.

**Table 9.**

Correlation coefficient ( $r$ ) between iEMG of vastus lateralis, biceps femoris, soleus, and blood pH, lactate, bicarbonate ion ( $\text{HCO}_3^-$ ), sodium ( $\text{Na}^+$ ) and potassium ( $\text{K}^+$ ).

	pH	Lactate	$\text{HCO}_3^-$	$\text{Na}^+$	$\text{K}^+$
iEMG of vastus lateralis	0.18*	0.02	-0.06	0.08	-0.08
iEMG of biceps femoris	0.40**	-0.39**	-0.30**	0.31**	0.24*
iEMG of Soleus	0.22**	0.04	0.14	0.17	-0.02

Notes: \* denotes significance at  $P<0.05$  level; \*\* denotes significance at  $P<0.01$  level.

#### 4.8.4 Power, respiratory variables and scales

A weak positive correlation ( $r=0.33$ ;  $P<0.01$ ) was found to exist between power output (Figure 13) and  $\dot{V}\text{O}_2$  (Figure 19) during the non-sprint stages of the two trials. A positive relationship ( $r=0.32$ ;  $P<0.01$ ) was also shown between power and RER (Figure 20) during the non-sprint stages. Perception of effort during both trials (Figure 24) was strongly correlated to perceived pain intensity (Figure 25) ( $r=0.88$ ;  $P<0.01$ ).

## **CHAPTER 5      DISCUSSION**

The purpose of this study was to investigate variables from numerous fatigue models concurrently to give more insight into understanding the fatigue process during prolonged endurance cycling in both hot (34°C) and cold (10°C) environmental conditions. Due to the prolonged and dynamic nature of the protocol used, this study also allowed us to examine contributors to fatigue during both high-intensity endurance exercise (1-km and 4-km sprints), as well as during prolonged endurance exercise (fatigue as the time trial progressed). Evidence for and against the various proposed models of fatigue outlined in Chapter 2 will be discussed.

### **5.1      Changes in Performance Variables in Hot and Cold Conditions**

The first hypothesis of this thesis was that cycling performance would be reduced in the hot versus cold climate as indicated by an increased performance time due primarily to the higher core temperature. Higher core temperatures have been shown to be related to a reduced central neural recruitment of skeletal muscle motor units in response to afferent sensory feedback (Cheuvront & Haymes, 2001; Kay & Marino, 2000; McArdle et al., 2001; Nybo & Nielsen, 2001a; Tucker et al., 2004; Wilmore & Costill, 1999). The central thermoregulatory model of fatigue (see Section 2.8.1) suggests that reductions in power output occur in response to temperatures in the brain reaching near critical levels (Cheuvront & Haymes, 2001; Kay & Marino, 2000; McArdle et al., 2001; Nybo & Nielsen, 2001a; Tucker et al., 2004; Wilmore & Costill, 1999). Findings from the present study do not support this hypothesis. The present study showed that iEMG and power output were decreased in the heat *before* core temperature reached critical levels (i.e., >40°C). This finding lends support to the notion that reductions in power output are not solely the result of an increased core (rectal) temperature acting on the brain and causing altered muscle recruitment patterns, as was previously believed (Cheuvront & Haymes, 2001; Kay & Marino, 2000; McArdle et al., 2001; Nybo & Nielsen, 2001a; Tucker et al., 2004; Wilmore & Costill, 1999).

Other findings from the present study, however, do support the thermoregulatory model of fatigue. For example, power output averaged during the non-sprint, 1-km, and 4-km sprints was significantly reduced in the hot trial when compared to the cold trial (all  $P < 0.01$ ; Table 3). Indeed, power output was significantly decreased in the hot condition during both sprint and non-sprint periods beyond 28-km (Figure 13). These results support the findings of Tucker et al. (2004) who showed that rectal temperature was not significantly different between hot ( $35^{\circ}\text{C}$ ) and cold ( $15^{\circ}\text{C}$ ) trials over the first 15-km of a 20-km time trial. Tucker et al. (2004) also found that power output was significantly reduced in the hot compared to the cold condition before significant differences in rectal temperature evolved. These authors hypothesized that because power output was highest during the final 5% of the time trial, when rectal temperature was also highest, that power output during the hot trial was reduced by an anticipatory response in the brain causing reductions in muscle recruitment. This hypothesis was supported by the finding of significant reductions in iEMG of vastus lateralis in the hot compared to the cold trial before significant increases in core temperature occurred (Tucker et al., 2004). In contrast, the present study found that iEMG of vastus lateralis was not significantly different between the two trials (Figure 2). This may have been due to the longer ride distance used in the present study (100-km), resulting in a much lower muscle activation of the lower limb during both trials. The much shorter protocol used by Tucker et al. (2004) (20-km) elicited vastus lateralis iEMG values that were nearly half of those found in the present study. Interestingly, a previous study using the same 100-km protocol as that of the present study in thermoneutral conditions ( $22^{\circ}\text{C}$ , 55% rh) found similar iEMG values, whereby ~20% or less of rectus femoris was active during the entire time trial (St Clair Gibson, Schabort et al., 2001). Certainly the shorter the prescribed competition or protocol, the greater the power output tends to be (Lucia, Hoyos, Santalla, Earnest et al., 2003; Ulmer, 1996). Muscle activation (iEMG) of biceps femoris and soleus was, however, significantly lower in the hot compared with the cold trial, and these differences began to evolve in biceps femoris after 22-km (Figure 15), and in soleus after only 15-km (Figure 16).

The iEMG of the lower limb and power output were both significantly different between the hot and cold trials, suggesting that the differences in power output between the two trials were mainly the result of differences in central

activation or neuromuscular propagation failure (Allman & Rice, 2002; Avela et al., 2001; Davis et al., 2000; Davis & Bailey, 1997; Green, 1997; Kay et al., 2001). As previously mentioned, Tucker et al. (2004) suggested that the reduction in central command despite the existence of any thermal stress (significant increases in rectal temperature) may be the result of an anticipatory response in the brain which reduces muscle activation to dampen the rate of increases in core temperature. If this theory is correct then it raises the question as to how the brain identifies the significant differences in environmental stress and alters muscle activation accordingly. Both mean body temperature and mean skin temperature were significantly higher in the hot compared with the cold trial (Figures 17b and 17c). Indeed, afferent sensory feedback from skin and muscle thermoreceptors may play a significant role in the control of muscle activation during exercise in the heat (Tucker et al., 2004). The involvement of peripheral sensory feedback influencing central command is supported in the present study by significantly higher perceived thermal sensations in the hot compared with the cold trial (Figure 23;  $P < 0.001$ ) and increased significantly with increasing distance in both the hot and the cold trials (both  $P < 0.01$ ). In fact, in the hot trial subjects perceived thermal sensation to be significantly increased from 5.1 (warm) at 5-km to 6.1 (hot) at 22-km, and this did not change significantly until the final sprint. In contrast to previous findings (Hunter, St Clair Gibson, Mbambo et al., 2002; Nybo et al., 2002; Nybo & Nielsen, 2001b), increases in perceived exertion during the hot trial did not parallel increases in core temperature, and suggests that perceived thermal sensation may be a better psychological (perceptual) indicator of thermal stress (core body temperature) than pain or RPE. Indeed, the significantly lower muscle activation in the hot trial found in the present study may be the result of peripheral thermoregulatory fatigue. Afferent sensory feedback from thermoreceptors located in the skin may be the cause of the reduced central drive from the brain and resulting reduction in power output. This reduction in muscle activation seems a logical safety mechanism during exercise in the heat as it would prevent a rapid rise in body temperature and prevent core temperature from reaching dangerous levels. In fact, rectal temperatures in both hot and cold trials rarely exceeded 40°C in most subjects, and no subjects reached values representative of heatstroke (41°C).

## **5.2 Relationship between Fatigue Variables and Power Output**

The second hypothesis of this study was that decreases in cycling intensity (i.e., decreased power output) during the 1-km and 4-km sprints would result in decreases in muscle activation of the lower limb, associated with increases in core temperature and perceived exertion. There were no significant differences in rectal or skin temperature during the 1-km or 4-km sprints compared to preceding distances in the same environmental condition. This occurred despite significant differences in power output in the 1-km and 4-km sprints compared to non-sprint stages in both the hot and cold trials. Moreover, power output was found to gradually decrease during the non-sprints in the heat, and during the 1-km and 4-km sprints in both the hot and cold conditions (Figure 13). Noticeably, this occurred in the cold condition even though rectal, mean body and mean skin temperatures were significantly lower than in the hot trial when compared to the cold trial (Figure 17). Thus, it is proposed that other models of fatigue (neuromuscular fatigue model; cardiovascular anaerobic model; energy supply/energy depletion model) and not the thermoregulatory model may be causal for the reduced power output that occurred during the cold trial, and especially in relation to the decline in power output with above-threshold intensities during the 1- and 4-km sprints in the cold condition. Support for these abovementioned models of fatigue will be discussed in Section 5.3.

One limitation of the present study was that muscle temperatures were not measured, and it is possible that increases in muscle temperature to critical levels may have been responsible for a reduced sprint power output (Ray & Gracey, 1997; Tucker et al., 2004). However, as subjects were able to significantly increase power output under both environmental conditions during the last sprint (99-km) in comparison to the sprint at 72-km (Figure 13), and none of the temperature measures changed significantly during the sprint stages (Figure 17), it is more probable that the reduction in power output with increasing distance was not the result of an increase in muscle temperature to critical levels.

A major advantage of the present study over previous research is that muscle activation of a number of muscles was recorded (St Clair Gibson, Schabort et al., 2001; Tucker et al., 2004). Therefore, it is more representative of the entire lower limb. It was found during the 100-km time trial that muscle activation (as measured



by iEMG in this study) of all three muscles investigated gradually declined under all conditions (Figures 14, 15 and 16). In fact, significant correlations were found to exist between iEMG of biceps femoris, soleus, and vastus lateralis during the non-sprints, 1-km and 4-km sprints (Table 8; all  $P < 0.01$ ). This suggests that there was not a change in the excitatory or inhibitory commands to some muscles in order to compensate for a fatiguing muscle as previously suggested (St Clair Gibson, Schabort et al., 2001; Tucker et al., 2004). In the present study, it was found that iEMG activity of vastus lateralis, biceps femoris and soleus declined with increasing distance during both the 1- and 4-km sprints in both hot and cold trials (Figures 14, 15 and 16). This decline in iEMG activity with prolonged exercise supports the findings of St Clair Gibson, Schabort and Noakes (2001), who found similar reductions in iEMG of rectus femoris during the 1-km and 4-km sprints when using the same 100-km cycling protocol; these decreases in muscle activation also paralleled reductions in power output. Such findings however, contradict other studies that have showed an increase in iEMG during fatiguing exercise (Bull et al., 2000; Hug et al., 2004). Such an increase in iEMG is thought to represent activation of a greater number of motor units in order to compensate for peripheral fatigue (Borrani et al., 2003; Bull et al., 2000; Hug et al., 2004). Unfortunately, cardio-respiratory measures were not measured during the sprints due to concerns that the mouth-piece apparatus might influence sprint performance. Thus, it is unknown whether  $\dot{V}O_2$  during the sprints actually increased as the trials progressed, as an increase in oxygen consumption might reflect an increase in the number of recruited fast twitch muscle fibres compensating for fatiguing slow twitch fibres (Borrani et al., 2003; Bull et al., 2000; Hug et al., 2004). There was however, no significant increase in  $\dot{V}O_2$  during the non-sprints in either the hot or cold trials suggestive that there was not an increase in activation of Type II muscle fibres with increasing exercise duration (Borrani et al., 2003; Hug, Laplaud et al., 2003).

The reduction in neuromuscular activity during the sprints in the present study suggests that in the majority of cases, fatigue during prolonged cycling may be caused by a reduction in central command. This 'central activation failure' may be the result of intracortical inhibition (Millet et al., 2003; Paasuke et al., 1999) or perhaps a response to afferent sensory feedback from peripheral organs (such as the muscles) (Ciubotariu et al., 2004) or both. Unfortunately, due to the nature of the

task in the present study, the use of twitch interpolation to determine the extent of the fatigue would not have been practical. It is therefore difficult to distinguish between a voluntary/psychological decline in central command (i.e. due to a lack of motivation and/or boredom) and physiological factors that may have caused the reduced central activation. The present study used an IT-RT assessment to assess the potential development of central fatigue in the brain (Burns & Nettelbeck, 2003). Interestingly, neither IT nor RT was significantly different between the hot and cold conditions, nor did IT or RT change significantly following the time trials (Table 6). This suggests that a 100-km time trial in either hot or cold conditions causes no significant change in the ability of a subject to process a visual stimulus (IT) (Nettelbeck, 2001) or to react to a visual stimulus (RT) (Burns & Nettelbeck, 2003), and suggests that central processing ability and motor drive were unaltered by the condition or the 100-km trial. One limitation to this finding, however, was that the IT/RT test was performed seven minutes following completion of the time trial, which could be enough time for a well-trained cyclist to recover from central fatigue.

It has been suggested that psychological factors play a significant role in the development of central activation during exercise (Borg, 1982; Cain & Stevens, 1973; Cochrane & Sleivert, 1999; Kayser, 2003; Tattersson et al., 2000). Nicol et al. (1991) found that both force development and iEMG decreased approximately 30% following a 42-km marathon. The authors concluded that this reduction in central command was due to a decline in conscious effort or perhaps a change in the central activation patterns. During such a prolonged protocol as in the present study, boredom may have played some role in the central recruitment strategy (Brooks et al., 2000b; Febbraio, 2000; Hargreaves & Febbraio, 1998; Nybo & Nielsen, 2001a; Tattersson et al., 2000). However, RPE gradually increased throughout both trials in the present study suggesting that subjects continued to exert a greater effort as the trial progressed despite gradual reductions in power output (Tucker et al., 2004). The findings of the present study confirm those of St Clair Gibson, Schabort, et al. (2001) who found that during prolonged cycling, iEMG declined despite a greater perception of effort. Indeed, a greater RPE indicates that a central activation failure, as seen in the present study, was not solely the result of a reduction in motivation as the protocol progressed (St Clair Gibson, Schabort et al., 2001). The reduction in central activation may have begun in the brain (Davis, 1995; Davis et al., 2000;

Davis & Bailey, 1997; Kay & Marino, 2000) or perhaps may be the result of afferent sensory feedback from receptors located in the muscles themselves (Hampson et al., 2001). Interestingly, perceived pain intensity in the quadriceps increased at similar rates in the hot and cold trials despite the significantly different power output and muscle activation between the two trials (Figure 25). Such increases in muscle pain have been found to coincide with reductions in muscle activation (Ciubotariu et al., 2004; Hampson et al., 2001) and thus supports the muscle trauma model of fatigue (see Section 2.6 for review). It could be speculated then that fatigue may be a response to pain receptors within the active muscles reacting to homeostatic imbalances, and that these receptors relay afferent sensory feedback to the motor cortex, resulting in a reduction in neural drive. If this theory is correct, then it is questionable as to how subjects are able to produce significantly greater amounts of force during the final sprint (99-km) compared to their 1-km sprint at 72-km, even with a greater perceived pain intensity in both hot and cold trials. The present study used a 'closed-loop' protocol whereby subjects were aware of the total distance they were asked to complete, the total number and distance of each sprint, and the positioning of each sprint. Thus, a greater power output during the final sprint is likely to be due to centrally controlled pacing strategies (Ulmer, 1996), whereby muscle activation and power output are controlled by a central controller (central governor model) in order to ensure that subjects could complete the task without ever reaching or being in danger of reaching complete exhaustion (Noakes, 2000a; Ulmer, 1996). As expected from previous studies, RPE gradually increased throughout both the hot and the cold trials (Figure 8) (St Clair Gibson, Schabort et al., 2001; Tucker et al., 2004) and was highly correlated to pain intensity ( $r=0.88$ ;  $P<0.01$ ). The fact that subjects only reached near maximal RPE during the final sprint ( $19.0 \pm 1.6$  and  $18.6 \pm 1.8$  Borg Units in the hot and cold trials, respectively; Figure 8) further supports the existence of a pacing strategy used in order to ensure that subjects are able to complete the trial without reaching complete exhaustion.

The question still remains as to what homeostatic imbalances are responsible for the increases in pain, reductions in central drive, and reductions in power output during the trials. It has long been felt that the generation of lactic acid in the muscles during prolonged high-intensity exercise is the primary mechanism for the reduction in exercise performance. The lactic acid produced during anaerobic glycolytic

conditions dissociates into lactate and hydrogen ions, and as a result the pH is lowered. It was previously felt that the reduced intramuscular pH may reduce exercise performance (Davis & Bailey, 1997; Stackhouse, Reisman et al., 2001) by decreasing glycolytic flux through the inhibition of phosphofructokinase (PFK), or by interrupting contractions by reducing  $\text{Ca}^{2+}$  release and removing  $\text{Ca}^{2+}$  from troponin (Brooks et al., 2000b; Hill et al., 2001; Stackhouse, Reisman et al., 2001), or by stimulating pain receptors (Brooks et al., 2000b; Hampson et al., 2001; Hill et al., 2001; Stackhouse, Reisman et al., 2001). Furthermore,  $\text{H}^+$  ions released into the blood may affect performance by influencing pain receptors in the brain (Bogdanis et al., 1994), inhibiting  $\text{O}_2$  transportation via haemoglobin (Brooks et al., 2000b), and by reducing the dissociation of free fatty acids into the blood (Brooks et al., 2000b). To the contrary, the present study showed that blood pH actually increased throughout both the hot and cold trials (Figure 18a) despite increases in RPE and perceived pain intensity (Figures 24 and 25). This shows that increases in perceived exertion and pain intensity during prolonged exercise are in fact not related to increases in the acidity of the blood as previously thought (Brooks et al., 2000b; Hampson et al., 2001). The increase in blood pH as found in the present study may have been related to an anticipatory overcompensation in response to the reduced pH that likely occurred during the sprints (suggested by the reduction in pH measured during the final sprint; Figure 18a). Certainly one of the limitations of the current study was that blood measures were not recorded during the majority of the 1-km or 4-km sprints to confirm this. Despite no significant increase in bicarbonate ion concentrations (Figure 18d) or blood lactate concentrations (Figure 18g) during the non-sprint stages, both blood bicarbonate ion ( $r=0.51$ ;  $P<0.01$ ) and lactate ( $r=-0.42$ ;  $P<0.01$ ) were correlated to pH. Another major limitation of this study was that muscle biopsies were not taken, so specific alterations in muscle biochemistry that might have contributed to fatigue remain unknown.

Blood glucose levels in the present study did not change significantly over distance in either the hot or the cold trials (Figure 18l), and this was expected as subjects consumed carbohydrate at a rate of  $60 \text{ g}\cdot\text{h}^{-1}$  during the trial. The capacity to oxidise carbohydrate, therefore, was likely not limited by the availability of blood glucose. Use of the same protocol, however, in thermoneutral conditions ( $\sim 20^\circ\text{C}$ ; 55% rh) showed muscle glycogen content to be significantly reduced from  $572\pm 107$

to  $96 \pm 63 \text{ mmol} \cdot \text{kg}^{-1} \text{ dry wt}$  (St Clair Gibson, Schabort et al., 2001). Similar rates of glycogen depletion likely occurred in the present study with an even greater carbohydrate depletion possible during the hot trial (Parkin et al., 1999). The fall in power output with increasing distance could be due to significant reductions in muscle glycogen stores (Brooks et al., 2000b). While blood glucose levels were not significantly reduced in either trial, RER slightly declined with increasing distance in both the hot and cold conditions (Figure 20;  $P < 0.05$ ), suggestive of an increased reliance on lipid metabolism for ATP resynthesis as the trial progressed (McArdle et al., 2001). Blood urea content also increased significantly as the trials progressed (Figure 18k;  $P < 0.05$ ), suggestive of an increase in protein metabolism as the trial progressed (McArdle et al., 2001). It is proposed that the reductions in power output during both trials in the present study may partially occur in response to afferent sensory feedback relaying information about reductions in muscle glycogen content (Energy Depletion Model; Section 2.3) to a central command post. Indeed, Davis et al. (2000) has suggested that nutritional status (especially that of carbohydrate) may effect neurotransmitters, causing a reduction in central drive. Although blood glucose was maintained throughout the trials, the reduction in power output during the higher intensity sprints may in fact be due to insufficient muscle glycogen available to supply the millisecond energy demands that would exist during the sprints (Shulman & Rothman, 2001).

Muscle activation may also have been reduced in response to changes in ionic gradients (Lepers et al., 2000; Lepers et al., 2002; Ray & Gracey, 1997; St Clair Gibson, Lambert et al., 2001; Walton et al., 2002) or perhaps altered  $\text{Ca}^{2+}$  homeostasis (Brooks et al., 2000b; Fowles et al., 2002; Green, 1997; Hamlin & Quigley, 2001b; McKenna et al., 1996; Stackhouse, Reisman et al., 2001). While  $\text{K}^+$  levels were not significantly different between trials, extracellular  $\text{Na}^+$  concentration was significantly lower in the hot versus the cold trial from 28-km to the end of the trial (Figure 18h). This is most likely due to a significantly greater change in body mass (i.e., sweat rate;  $P < 0.01$ ) and thus sodium loss during the hot trial (Table 5). The lower extracellular  $\text{Na}^+$  concentration found in the hot trial may have been responsible for alterations in muscle membrane excitability and reduced central activation (Green, 1997; Oba et al., 2000). Indeed, in the present study a weak correlation ( $r = 0.31$ ;  $P < 0.001$ ) was found to exist between blood  $\text{Na}^+$  concentration

and iEMG of biceps femoris. It is interesting to note that this difference in  $\text{Na}^+$  concentration occurred despite the supplementation of 340 mg  $\text{Na}^+$  per hour supplied in the carbohydrate-electrolyte beverage. The potential implication for this finding is that athletes who perform exercise in conditions of high temperature and humidity may benefit from an additional supplementation of sodium to their carbohydrate electrolyte beverage, and future research should examine this premise. Despite significantly lower  $\text{Na}^+$  concentrations under hot versus cold conditions, muscle biopsies were not taken in the present study, so alterations in contractile proteins, as well as the regulation of intracellular potassium and calcium are unknown. Such chemical imbalances have the potential to alter muscle membrane excitability, as well as the potential to influence neuromuscular/afferent sensory pathways to cause alterations in muscle recruitment patterns (Hampson et al., 2001).

### **5.3 What is the Cause of Fatigue during Prolonged Cycling in Hot versus Cold Conditions?**

#### **5.3.1 Hot condition**

The third hypothesis of this study was that decrements in power output in the hot condition would be the result a teleoanticipatory reduction in muscle activation of the lower limb due to rises in core temperature. This study has provided support for the concept that the fatigue process during prolonged cycling is multifactorial and highly task specific (Hunter et al., 2004). As mentioned, the most predominant factors relating to fatigue during prolonged cycling in the heat (34°C) appear to be due to aspects associated with the thermoregulatory model of fatigue. Significantly higher mean skin and core temperatures appear to cause a ‘teleoanticipatory’ reduction in central drive in order to prevent heat exhaustion (Tucker et al., 2004). A major advantage of the present study over previous research is that multiple physiological, biomechanical and psychological factors were examined concurrently. Unfortunately these measures neither contradicted nor offered a better explanation for fatigue than has been previously stated. These findings will be discussed below.

As expected, blood  $\text{PCO}_2$  was significantly higher in the cold trial compared to the hot trial due to an increased exercise intensity (Figure 13) and higher

metabolic rate achieved in the cold trial, and is supported by the significantly greater oxygen consumption ( $P<0.05$ ) and minute ventilation ( $P<0.01$ ) measured in the cold trial (Figures 19 and 21). Interestingly, heart rate was not significantly different under hot or cold conditions (Figure 22) (Cochrane & Sleivert, 1999; Green, 1997; Nielsen et al., 1993; Nybo & Nielsen, 2001b). While one might expect the higher metabolic rate achieved in the cold trial to increase heart rate above that found in the hot trial, the higher levels of core body temperature, which are known to increase circulating levels of catecholamines, would likely increase heart rate to those levels achieved in the higher intensity cold trial (Tucker et al., 2004). The finding that heart rate was similar between both hot and cold trials lends support to the cardiovascular/anaerobic model of fatigue, as heart rate may have been a key limiting factor to exercise performance (Delp & Laughlin, 1998; Radegran et al., 1999). Indeed, the reduction in power output seen in the hot trial might have been due to an increase in skin blood flow (Hunter, St Clair Gibson, Mbambo et al., 2002; Nybo et al., 2001) and a resulting decrease in muscle blood flow (peripheral thermoregulatory fatigue) (Hunter, St Clair Gibson, Mbambo et al., 2002; 1998). On the other hand, Figure 22 shows that heart rate only reached ~85-90% of its maximum during the final sprint, and demonstrates that considerable heart rate reserve remained despite a maximal perceived exertion. This suggests that heart rate (and more importantly cardiac output) may limit performance during prolonged exercise, as heart rate did not increase to levels obtained during the  $\dot{V}O_{2max}$  test.

Rating of perceived exertion in the present study was not significantly different between hot and cold trials, and reached near maximum values by the end of the trial (St Clair Gibson, Schabort et al., 2001; Tucker et al., 2004). This suggests that in both trials, subjects were able to pace themselves in order to complete the trial without reaching complete exhaustion (Ulmer, 1996), which further supports the existence of an anticipatory control of power output in order to ensure that subjects are able finish the trial with the best possible performance (Ulmer, 1996). An unfortunate limitation to the findings in the present study was that despite controlling for physical exertion in the week prior to each trial (exercise logbooks) perceived physical fatigue (MFI-20) was significantly greater prior to the hot trial, compared to the cold ( $P<0.05$ ; Table 3). Nevertheless, this should be considered to be a strength of the current study as previously similar studies have not controlled for such

psychological indices of fatigue. This could also be an indication of the effects that teloanticipation plays on psychological fatigue, as subjects were aware of what climatic condition they would be performing in prior to completing the questionnaire. Nevertheless, motivation was not significantly different prior to the trials (Table 3).

The findings from the present study suggest that the most predominant cause of fatigue during prolonged cycling in the heat would seem to be related to an anticipatory reduction in central command. Reductions in power output during exercise in the heat are likely to be due to a significantly greater skin and whole body temperature causing altered afferent sensory feedback, increased perceived thermal sensation and thus a reduced drive from central command. Further work is needed to examine the involvement of thermo-receptive afferent sensory feedback on central command to verify this conjecture.

### **5.3.2 Cold condition**

The third hypothesis of the present study was also that decrements in cycling power output would be associated with reduced blood pH and glucose causing a reduction in muscle activation of the lower limb. Indeed, the most contributing factor to fatigue in the cold trial does not appear to be the same as that found in the hot trial (thermoregulatory fatigue model). Power output during the non-sprint portion of the cold trial remained statistically unaltered, but was reduced significantly during the sprint portions of the cold trial (Figure 13). Thus, fatigue during high-intensity cycling in the cold did not appear to be due to an increase in acidity of the blood nor due to a reduction in blood glucose as has been traditionally believed (Brooks et al., 2000b). As with the hot trial, it would appear that the most dominant factor reducing power output during prolonged cycling in the cold was due to a centrally controlled reduction in muscle activation (iEMG). Central fatigue, as seen in the cold trial irrespective of significantly lower rectal, mean skin, mean whole body temperature and perceived thermal sensation suggests that a very different process is responsible for the altered neural recruitment patterns. As previously mentioned, the reduction in central recruitment during exercise in the cold may be linked to afferent sensory feedback relaying significant reductions in carbohydrate stores (muscle glycogen). Also, at no time during either trial did oxygen consumption reach values indicative



of the subjects  $\dot{V}O_{2\max}$  even during the final sprint where  $\dot{V}O_2$  was less than 85% of  $\dot{V}O_{2\max}$ . This suggests that the fatigue during prolonged exercise in the cold may be limited by the delivery of oxygen to the working muscles and thus supports the cardiovascular/anaerobic model of fatigue. Indeed, heart rate can be seen to parallel  $\dot{V}O_2$  (Figures 19 and 22) and reached similar peak values during the final 1-km sprint. Moreover,  $\dot{V}O_2$  was found to partially correlate with decrements in power output ( $r=0.33$ ;  $P<0.01$ ), suggesting that oxygen delivery may have partially influenced exercise performance. If oxygen supply does restrict performance then the question is raised as to why subjects are not able to increase heart rate to maximum levels during the final sprint. Despite having no significant change in body mass (Table 5), cardiovascular fatigue may have been caused by a fluid compartment shift (extracellular fluid shifting intracellularly or into the interstitial compartments) causing fluid loss from the cardiovascular system. Blood hematocrit levels remained statistically constant throughout both trials, and not significantly different between the hot and cold trials (Figure 18m), suggesting that hydration status throughout both trials remained adequate.

## CHAPTER 6      CONCLUSION AND RECOMMENDATIONS FOR FURTHER RESEARCH

### 6.1      Conclusion

In conclusion, it was found that differing physiological, biomechanical, environmental, mechanical, and psychological factors influence fatigue in hot compared to cold environments. Existing models of fatigue have accredited a reduction in force or power development to be a result of a homeostatic imbalance of one or more systems that are vital for exercise performance. It seems from this study that reductions in power output during exercise in the heat are closely related to an anticipatory reduction in central recruitment (from the brain) caused by an irregular elevation in mean skin and whole body temperature and not increases in core temperature. This was because power output and muscle activation in the hot trial were found to be reduced *before* significant rises in core temperature occurred. This study found that prolonged cycling in the heat was associated with a greater perceived thermal sensation, mean whole body and mean skin temperatures. Muscle iEMG activation of the lower limb was significantly lower in the hot trial at 22-km (biceps femoris) and 15-km (soleus) despite no difference in core body temperature, perception of effort, pain intensity, RER, or heart rate. It is therefore likely that the significantly lower power output during exercise in the heat, as seen in the present study, is due to a centrally controlled reduction in muscle activation in order to limit the rate at which core temperature rises. It is postulated that this altered central recruitment is in response to afferent sensory feedback from thermoreceptors located within the skin and/or muscles. Further reductions in power output during prolonged cycling in the heat may also be due to a reduced membrane excitability as caused by a gradual decrease in extracellular  $\text{Na}^+$  concentration due to sweat loss. This was found despite the ingestion of  $\text{Na}^+$  at the rate of  $340 \text{ mg}\cdot\text{h}^{-1}$ .

A decline in power output during exercise in the cold would also seem to be due to a reduction in central command. 1-km and 4-km sprint power outputs gradually declined in the cold trial despite a lower perceived thermal sensation, mean skin, whole body and core body temperatures. 'Central activation failure' as seen in the cold trial appears more closely related to afferent sensory feedback responsive to changes within the muscle itself. Interestingly, fatigue during prolonged cycling in the cold was not closely associated with an increase in blood lactate, or a reduction in blood pH or blood glucose. The most influential factor causing fatigue during the sprints in the cold could not be determined from the present set of measurements. It is however, proposed that reduced muscle glycogen content, altered membrane excitability or perhaps increases in pain intensity may have been responsible for afferent sensory feedback during the sprints causing a reduction in central activation. Alternatively, performance during the 1-km and 4-km sprints may have been limited by the ability of the cardiovascular system to supply sufficient blood (oxygen) to the working muscles, as  $\dot{V}O_2$  and heart rate only reached 85-90% of maximum values achieved during the  $\dot{V}O_{2\max}$  test.

## 6.2 Recommendations for Further Research

On the basis of findings from the present study it is believed that future research examining fatigue during prolonged cycling in both hot and cold conditions should concurrently examine a greater number of physiological systems. In particular, further research should include examination of afferent sensory feedback, the origination of central command and the relationship between the two. Investigations into afferent sensory feedback during prolonged exercise in the heat should focus on the activation of thermoreceptors located within the body (in particular skin and muscles thermoreceptors). Future research examining the effects of prolonged exercise on fatigue should further address possible biochemical and structural changes that occur within the muscles themselves through the use of muscle biopsies, MRI, and muscle temperature thermistors.

A further recommendation for future research is the investigation of the effects of sodium loss and supplementation during prolonged cycling in the heat. Despite the ingestion of an electrolyte-carbohydrate drink during the time trials,

sodium declined during exercise in the heat. Reduced levels of extracellular sodium (hyponatremia) have the potential to limit exercise performance under hot and humid environmental conditions. It is further postulated that although body mass did not change significantly in either the hot or cold trials, performance may be affected by alterations in plasma volume and cellular hydration caused by prolonged exercise in both the hot and cold conditions. Further research is needed to examine how performance can be further optimized through appropriate fluid and electrolyte consumption.

The development of pain during prolonged exercise and its relationship to perception of effort also requires further research. An interesting discovery in the present study was that blood pH increased as pain intensity increased. Perhaps pain receptors are activated through alkalytic changes in blood pH? Regardless, future research needs to address factors that may cause pain during fatiguing exercise and whether relationships exist between the development of pain, afferent sensory feedback, and reductions in central activation.

As there was no evidence for differences between inspection time and reaction time in the present study, future research is also required to determine a more appropriate assessment of a possible central fatigue during exercise. An improved assessment of IT/RT during exercise may provide us insight into the processes of visual interpretation and reaction time when in a fatigued state.

## CHAPTER 7      REFERENCES

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# Fatigue in Cycling Warm Vs Cool Climates

Research is expected to commence in June/July 2004 at the exercise physiology climatic chamber, Edith Cowan University, Joondalup in an attempt to better understand the physiology behind fatigue in cycling in cool Vs warm environments.

This study will involve;

- VO<sub>2</sub> max assessment
- Lactate, ventilation and anaerobic threshold determination
- Quadriceps, hamstring and calf muscle strength tests
- 100 km performance time trials in warm and cool climates

Athletes will be required to report to the laboratory on four separate occasions;

- VO<sub>2max</sub>, threshold assessments and strength tests
- Familiarisation test (100-km time trial at 22° C)
- Performance time trial (100-km time trial at either 10° C or 34° C)
- Performance time trial (100-km time trial at either 10° C or 34° C, the opposite performed in session 3)

Athletes will receive a fitness appraisal and be educated as to the optimum training patterns to avoid fatigue and attain peak performance.

Testing is limited to athletes who are;

- Male
- 18-45 years old
- Riding in excess of 250 km/week

If you are interested contact Chris on:  
0404 299 331 or [c\\_abbiss@ecu.edu.au](mailto:c_abbiss@ecu.edu.au)

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Edith Cowan University,  
Joondalup



**FATIGUE DURING PROLONGED CYCLING****EDITH COWAN UNIVERSITY****SCHOOL OF BIOMEDICAL & SPORTS SCIENCE****TRAINING LOGBOOK**

Name \_\_\_\_\_

**WEEK 1**

Date/day	Activity	Duration (distance/time)	Average speed	Intensity (average heart rate)

**WEEK 2**

Date/day	Activity	Duration (distance/time)	Average speed	Intensity (average heart rate)



### WEEK 3

Date/day	Activity	Duration (distance/time)	Average speed	Intensity (average heart rate)

### WEEK 4

Date/day	Activity	Duration (distance/time)	Average speed	Intensity (average heart rate)

### WEEK 5

Date/day	Activity	Duration (distance/time)	Average speed	Intensity (average heart rate)

## FATIGUE DURING PROLONGED CYCLING

EDITH COWAN UNIVERSITY

SCHOOL OF BIOMEDICAL & SPORTS SCIENCE

### DIETARY STANDARDISATION INSTRUCTIONS

PREPARED BY PROFESSOR LOUISE BURKE,

DEPARTMENT OF SPORTS NUTRITION, AIS

In this study, we will be investigating factors effecting fatigue during prolonged cycling. To do this effectively, we need to reduce the “day to day variability” in cycling performance that might otherwise mask small, alterations in exercise performance. One tactic is to standardise all the conditions under which trials are performed – including dietary preparation. Important factors include:

- Amount of carbohydrate and energy eaten during the 24 hours before the trial
- Fluid intake on the day before and morning of the trial
- Pre-trial meal

These instructions will help you to achieve a similar preparation for each trial

#### **Carbohydrate and fluid goals**

Aim: we want you to eat at least 6 g of carbohydrate per kg of your body weight on the day before each trial, and the same pre-trial meal on the morning of your trial (providing at least 1 g carbohydrate/kg). We also want you to consume at least 2 litres of fluid on this day (including all drinks consumed at meals or during training), and 400 ml of fluid at the meal consumed just before the trial

Steps:

1. Fill in your name..... and  
current body weight?.....kg
2. Calculate your carbohydrate intake (minimum) for the day before the trial:  
 $6 \times \text{BM} = \dots\dots\dots\text{g}.$

Calculate your carbohydrate intake (minimum) for the last meal, eaten 2  
hours before you start the trial:  $1 \times \text{BM} = \dots\dots\dots\text{g}.$

3. Keep a food record for the day before your first trial, concentrating on the carbohydrate-rich foods found in the table over the page, and the amount of fluid consumed. Use the table on the following page to add up how much carbohydrate is eaten at each meal or snack. Aim for the targets of at least 6 g/kg and at least 1 l/kg. Each of these “blocks” of food provides approximately 50 g of carbohydrate. It is not necessary to eat a whole block, or round numbers of blocks. Try to keep count in terms of quarter or half blocks
4. Once you have completed the first day’s record, this sets the amount that you need to eat for the next trials. It is simplest to try to repeat a very similar meal pattern for each of these days – i.e. stick to the same type and amount carbohydrate foods. If this is impractical, use the carbohydrate counter to replace one carbohydrate food with the amount of another carbohydrate choice that provides a similar amount of carbohydrate.

**Example**, on day one you might have eaten 2 rounds of cheese and salad sandwiches (4 thin slices of bread) for lunch, with a Juice Popper (unsweetened orange juice). The carbohydrate counter tells you that this is equal to 1 block (50 g carbohydrate) for the bread and just under a half block (or about 20 g of carbohydrate) for the Popper. If you want to swap the lunch menu, this same amount of carbohydrate could be found in 2 english muffins (with a similar kind of filling) and one carton of low fat flavoured yoghurt.

5. Keep a record of each day’s food intake so that we can check how well you were able to duplicate your carbohydrate intake and fluid intake for the next trials.
6. Repeat the same process for the meal eaten ~ 2 hours before the trial

## Ready reckoner of 50 g carbohydrate serves from common foods

Professor Louise Burke, Australian Institute of Sport

### CEREALS

Wheat biscuit cereal (e.g. Weetbix)	60g (5 biscuits)
'Light' breakfast cereal (e.g. Cornflakes, Weeties)	60 g (2 cups)
'Muesli' flake breakfast cereal (e.g. Sustain)	65 g (1.5 cups)
Toasted muesli	90 g (1 cup)
Porridge - made with milk	350 g (1.3 cups)
Porridge - made with water	550 g (2.5 cups)
Rolled oats	90 g (1 cup)
Cereal bar	2.5 x 30 g bar, 3 x 25 g bar
Rice cakes	6 thick or 10 thin
Rice, boiled	180g (1 cup)
Pasta or noodles, boiled	200 g (1.3 cups)
Canned spaghetti	440 g (large can)
Crispbreads and dry biscuits	6 large or 15 small
Fruit filled biscuits	5
Plain sweet biscuits	8-10
Cream filled/chocolate biscuits	6
Bread	110 g (4 slices white or 3 thick wholegrain)
Bread rolls	110 g (1 large or 2 medium)
Pita and lebanese bread	100 g (2 pita)
Chapati	150 g (2.5)
English muffin	120 g (2 full muffins)
Crumpet	2.5
Cake-style muffin	115 g (1 medium)
Pancakes	150 g (2 medium)
Scones	125 g (3 medium)
Iced fruit bun	105 g (1.5)
Croissant	140 g (1.5 large or 2 medium)
Rice-cream or creamed rice	330 g (1.5 cups)

### FRUIT

Fruit crumble	1 cup
Fruit packed in heavy syrup	280 g (1.3 cups)
Fruit stewed/canned in light syrup	520 g (2 cups)
Fresh fruit salad	500 g (2.5 cups)
Bananas	2 medium-large
Mangoes, pears, grapefruit and other large fruit	2-3
Oranges, apples and other medium size fruit	3-4
Nectarines, apricots and other small fruit	12
Grapes	350 g (2 cups)
Melon	1,000 g (6 cups)
Strawberries	1,800 g (12 cups)
Sultanas and raisins	70 g (4 Tbsp)
Dried apricots	115 g (22 halves)

## VEGETABLES AND LEGUMES

Potatoes	350g potato (one very large or 3 med)
Sweet potato	350 g (2.5 cups)
Corn	300 g (1.2 cups creamed corn or 2 cobs)
Green Beans	1,800 g (14 cups)
Baked beans	440 g (1 large can)
Lentils	400 g (2 cups)
Soy beans and kidney beans	400 g (2 cups)
Tomato puree	1 liter (4 cups)
Pumpkin and peas	700 g (5 cups)

## DAIRY PRODUCTS

Milk	1 liter
Flavored milk	560 ml
Custard	300 g (1.3 cup)
'Diet' yogurt and natural yogurt	800 g (4 individual tubs)
Flavored non-fat yogurt	350 g (2 x 200 g individual tubs)
Ice-cream	250 g (5 scoops)

## SUGARS AND CONFECTIONERY

<i>Sugar</i>	<i>50 g</i>
Jam	3 Tbsp
Syrups	4 Tbsp
Honey	3 Tbsp
Chocolate	80 g
Mars Bar (~ 60 g bar)	1.5 bars
Jelly confectionery	60g

## MIXED DISHES

Pizza	200 g (medium - 1/4 thick or 1/3 thin)
Hamburgers	1.3 Big Macs
Lasagna	400 g serve
Fried rice	200 g (1.3 cups)

## DRINKS

Fruit juice - unsweetened	600 ml
Fruit juice - sweetened	500 ml
Cordial	800 ml
Soft drinks and flavored mineral water	500 ml
Fruit smoothie	250-300 ml

## SPORTS FOODS

Sports drink	700 ml
Carbohydrate loader supplement	250 ml
Liquid meal supplement	250-300 ml
Sports bar	1-1.5 bars
Sports gels	2 sachets
Glucose polymer powder	60 g

**TRIAL 1: DAY BEFORE****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
Breakfast			
Lunch			
Dinner			
Snacks			

Total carbohydrate (AIM = g).....

Total fluid (aim = &gt; 2000 ml) .....

**TRIAL 1: MORNING OF TRIAL – LAST MEAL (2 HOURS PRE TRIAL)****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM =      g	AIM = 400 ML

Note: if you have a late morning/early afternoon trial, you may choose to eat an early breakfast, followed by this last meal. If so, please record the breakfast and repeat for all subsequent trials

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM =      g	

**TRIAL 2: DAY BEFORE****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
Breakfast			
Lunch			
Dinner			
Snacks			

Total carbohydrate (AIM = g).....

Total fluid (aim = &gt; 2000 ml) .....



**TRIAL 2: MORNING OF TRIAL – LAST MEAL (2 HOURS PRE TRIAL)****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM = g	AIM = 400 ML

Note: if you have a late morning/early afternoon trial, you may choose to eat an early breakfast, followed by this last meal. If so, please record the breakfast and repeat for all subsequent trials

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM = g	

**TRIAL 3: DAY BEFORE****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
Breakfast			
Lunch			
Dinner			
Snacks			

Total carbohydrate (AIM = g).....

Total fluid (aim = &gt; 2000 ml) .....

**TRIAL 3: MORNING OF TRIAL – LAST MEAL (2 HOURS PRE TRIAL)****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM = g	AIM = 400 ML

Note: if you have a late morning/early afternoon trial, you may choose to eat an early breakfast, followed by this last meal. If so, please record the breakfast and repeat for all subsequent trials

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM = g	

**TRIAL 4: DAY BEFORE****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
Breakfast			
Lunch			
Dinner			
Snacks			

Total carbohydrate (AIM = g).....

Total fluid (aim = &gt; 2000 ml) .....

**TRIAL 4: MORNING OF TRIAL – LAST MEAL (2 HOURS PRE TRIAL)****date:****name.....**

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM = g	AIM = 400 ML

Note: if you have a late morning/early afternoon trial, you may choose to eat an early breakfast, followed by this last meal. If so, please record the breakfast and repeat for all subsequent trials

Meal	FOOD AND DRINKS	CALCULATION OF CARBOHYDRATE CONTENT	CALCULATION OF ML OF FLUID CONSUMED
		AIM = g	

**FATIGUE DURING PROLONGED CYCLING**

EDITH COWAN UNIVERSITY

SCHOOL OF BIOMEDICAL &amp; SPORTS SCIENCE

**SUBJECT INFORMATION FORM****Summary**

This study will investigate the physiological responses of the body to prolonged endurance cycling in warm and cool climates. The purpose of this study is to assess the validity of developed fatigue models, thus, allowing us to better understand the mechanisms involved in fatigue during prolonged cycling in these climates. This research project has been approved by the Ethics Committee of Edith Cowan University.

**If I choose to participate, what will I have to do?**

As a participant in this study you will be asked to report to the laboratory on four separate occasions spaced at least seven days apart. In order to limit any factors that may lead to variation in “day to day” cycle performance you will be asked to complete a training/exercise log for the week prior to each trial and dietary log detailing dietary intake during the 24 hours prior to each trial. To provide you with sufficient carbohydrate prior to each trial you will also be educated on the recommended intake and foods necessary to obtain this. You will be asked to abstain

from cycling 24 hours prior to each test and you will be requested to engage in the following exercise:

1. Laboratory Based  $\text{VO}_{2\text{max}}$  Test – Cycle on a stationary bike at an intensity of 100W for 5 minutes. After this initial 5 minutes the intensity will increase 50W every 5 minutes until voluntary exhaustion. This test will be performed in standard laboratory condition (18-22°C). Results from this test will be used to establish how fit you are and identify the general fitness characteristics of the group. Toward the end of the test you will be asked to place a mouthpiece in your mouth. This mouthpiece is fed to a gas analyser which will continually measure your inspired and expired air. During the  $\text{VO}_{2\text{max}}$  test a small amount of blood will also be obtained via a small and sterile finger prick needle taken from the fingertip at 5 minute intervals. This blood will then be used to determine your blood lactate level during exercise.

2. Height, Mass and Sum of nine Skinfolds – These measurements will be performed to characterise the group and to determine your body composition.

3. Maximal strength test – Maximal isometric (stationary) contraction of the quadriceps, hamstring and calf muscles. During this test surface electromyography (EMG) electrodes will be placed on your skin to measure your muscular activity when you perform the contractions.

4. Laboratory Based 100-km Time Trials in the Hot and Cold – You will be asked to perform a series (n=3) of maximal effort 100-km time trials on a cycle ergometer in a climatic chamber set at 22°C (familiarisation), 10°C (Cold) and 34°C (Hot). During this time trial you will be asked to use a number of different sensors to assess your physiological response to the effort. Monitors will include: 1) a heart rate monitor, 2) skin thermocouples for assessing skin temperature, 3) a rectal probe, and 4) EMG electrodes for assessing muscle activity. Interspersed throughout the

trial you will be asked to evaluate your perception of effort and your thermal comfort. On six occasions researchers will also collect a small amount of blood from your finger tip to quantify your blood lactate level. Your oxygen consumption will be briefly assessed 11 times during the trial.

### **What are the Potential Risks and Discomforts?**

You will need to be prepared for EMG by shaving the surface of your skin at the three sites of placement. Surface EMG, gas analysis and skin temperature measures pose no risk to you.

**Exercise in the Heat** - You will be asked to ride on a stationary cycle ergometer as fast as you can for 100 km in the heat (34°C). As a competitive cyclist it is very likely that you have competed in hot races and although it is very unlikely that a cyclist will develop a heat illness during a self-paced time trial in warm conditions it is possible. For this reason we are putting some precautions in place including:

- 1) one of the researchers conducting the heat trials will have a current advanced first aid certificate and will know local health emergency procedures,
- 2) you will be excluded from participation if researchers feel you are not in good shape or have a history of heat exhaustion or heat stroke,
- 3) your power output (intensity) during performance trials will be self-selected by you allowing for exercise intensity to drop if there are any problems and,



- 4) you will be asked to report to the laboratory for testing in a well-hydrated state.

**Exercise in the cold** – You will be asked to perform a 100 km time trial in the cold (10°C). It is possible that you may find this slightly uncomfortable as you will be asked to refrain from wearing excess clothing (i.e. arm-warmers). However, as a competitive cyclist it is very likely that you have performed under such conditions and shortly after the onset of exercise you will rapidly warm-up.

**Body Temperature** – During your performance trials you will be using sensors that allow researchers to monitor your body temperature. You will be asked to use a rectal thermocouple. This sensor is a small thin Teflon coated probe that after detailed instruction will need to be self-inserted 10 cm past the anal sphincter. Rectal probes have been used to study various aspects of heat exposure for many years and the risks associated with use of these sensors are minor. Some individuals say that they feel uncomfortable once the rectal probe is inserted, however, in nearly all cases this discomfort passes once the subject begins exercising. Probably the most common complaint associated with using a rectal probe is embarrassment. Researchers will be sensitive to your situation and will be quick to answer any questions associated with the use of rectal probes. You will also have skin thermocouples taped to your skin but there is really no risk associated with this procedure.

**Blood Samples** – You will have up to six fingertip blood samples collected each session. A small needle will be used to pierce the skin prior to collecting the blood samples. Although the discomfort associated with this procedure varies, most athletes feel that this blood sampling technique is relatively minor. Although the use of finger prick blood analysis is a standard clinical procedure, there is a small risk

involved. Moreover, researchers performing this technique will be very proficient and experienced. There is a risk that you could develop bruising on the tips of your fingers and that your fingers will be sensitive for 2-5 days after the blood draw. Risk of infection is extremely low and will be made even lower because sterile techniques will be incorporated (rubber gloves, alcohol, sterile needles).

**Maximal Tests** –There are a number of slight risks associated with maximal exercise tests including light-headedness, fainting, abnormal blood pressure, chest discomfort, leg cramps, nausea, and in very rare cases, cardiac arrest. However, as a competitive cyclist these risks are minimal.

### **Will My Data Be Treated as Confidential?**

All personal information and test results recorded will remain confidential and will not be used for any purpose other than the current study. Moreover, all reports and presentations will not include your name or information that may identify you specifically as a subject.

No direct comparisons between other subjects participating in the study will be made at any stage of the testing. Analysis of data will be made on a group basis with means and variance of the whole group being compared between the temperatures. You are therefore not in competition with any other individuals in the study and will in no way be made to feel that your results are inadequate or poor.

### **Once I Commit to This Study Can I Quit?**

Yes, you will be free to withdraw from this study at any stage and for any reason without prejudice. One of the most important aspects of any research working

with human subjects is that as a subject you are fully informed of what you are being asked to do and that you know that you can quit at any time and not suffer any penalties. Your involvement is voluntary. You should not feel like you have been pressured or forced to participate. It is also important that you feel that all of your questions or concerns have been adequately addressed. Once you feel like you are fully informed about the methods and potential risks and discomforts associated with this study you will then be given the opportunity to sign an informed consent form. By signing this form you are letting researchers know that you are a voluntary informed participant. You are also indicating that you understand that you can remove your involvement at any time.

### **Requirements**

You will not be required to supply anything for the study. Should you have any questions relating to any of the information provided above, please feel free to contact either myself or Dr. Paul Laursen (6304 5012) for further explanation. If you have any concerns about this research, or would just like to speak to an independent person, you may contact Professor Robert Newton on telephone 9400 5711.

Yours Sincerely,

Chris Abbiss

School of Biomedical and Sports Science, Edith Cowan University

100 Joondalup Drive, Joondalup WA 6027

Phone: 0404 299 331 E-mail: [c\\_abbiss@hotmail.com](mailto:c_abbiss@hotmail.com)

## DOCUMENT OF INFORMED CONSENT

### Examination of Multidisciplinary Models during Prolonged Endurance Cycling In Warm Vs Cool Climates

I \_\_\_\_\_ have read the Subject Information Letter to Participants provided by the researcher and hereby agree to participate as a volunteer in a scientific investigation performed at Edith Cowan University.

The investigation and my part in the investigation have been defined and fully explained to me and I understand the explanation. A copy of the procedures of this investigation and a description of any risks and discomforts has been provided to me and has been discussed in detail with me.

- I have been given an opportunity to ask whatever questions I may have had and all such questions and inquiries have been answered to my satisfaction.
- I understand that I am free to deny any answers to specific items or questions in interviews or questionnaires.
- I understand that I am free to withdraw consent and to discontinue participation in the project or activity at any time.
- I understand that any data or answers to questions will remain confidential with regard to my identity.
- I certify to the best of my knowledge and belief, I have no physical or mental illness or weakness (i.e., previously suffered from heat exhaustion or heat stroke) that would increase the risk to me participating in this investigation.
- I agree that the research data obtained from this study may be published, provided I am not identifiable in any way.

Participant \_\_\_\_\_ Date \_\_\_\_\_

I, the undersigned, was present when the study was explained to the subject/s in detail and to the best of my knowledge and belief it was understood.

Investigator \_\_\_\_\_ Date \_\_\_\_\_

**FATIGUE DURING PROLONGED CYCLING**  
**EDITH COWAN UNIVERSITY**  
**SCHOOL OF BIOMEDICAL & SPORTS SCIENCE**  
**MEDICAL QUESTIONNAIRE**

Name: \_\_\_\_\_ Age: \_\_\_\_\_yr Weight: \_\_\_\_\_kg Height: \_\_\_\_\_cm

Briefly describe the type and amount of exercise you do.

Type:

\_\_\_\_\_

Amount:

\_\_\_\_\_

Do you smoke? YES NO

Have you smoked in the past? YES NO

Have you every been diagnosed with –

being overweight? YES NO

high blood pressure? YES NO

diabetes? YES NO

asthma? YES NO

any bleeding disorders? YES NO

Do you have any reason to believe that you are more at risk of cardiovascular disease than a normal member of the population of the same age and sex?

YES NO

If YES please give details

\_\_\_\_\_

\_\_\_\_\_

Have you ever had rheumatic fever?

YES NO

If YES please give details

\_\_\_\_\_

\_\_\_\_\_

Have you ever experienced heat exhaustion or heat stroke?

YES NO

If YES please give details

---

---

Have you ever suffered an injury during cycling and/or withdrawn from a race for any reason?

YES NO

If YES please give details

---

---

Is there anything that you are aware of that may limit your capacity to exercise? (e.g., Chronic back pain and/or other joint pain, severe headaches?)

YES NO

If YES please give details

---

---

Do you have any allergies?

YES NO

If YES please give details

---

---

Are you currently on any prescribed or non-prescribed medications?

YES NO

If YES please give details

---

---

Have you suffered from any viral infections, chronic tiredness or donated blood in the past two months?

YES NO

If YES please give details

---

---

Have you suffered from haemorrhoids, now or in the past?

YES NO

If YES please give details

---

Do you have any other complaint or any other reason that you know of which you think may prevent you from participating in and completing this experiment?

YES NO

If YES please give details

---

**I believe that the information that I have supplied is true and correct.**

**Print Name**

**Signed**

**Date**

---

# THERMAL SENSATIONS SCALE

**0.0 Unbearably Cold**

**0.5**

**1.0 Very Cold**

**1.5**

**2.0 Cold**

**2.5**

**3.0 Cool**

**3.5**

**4.0 Comfortable**

**4.5**

**5.0 Warm**

**5.5**

**6.0 Hot**

**6.5**

**7.0 Very Hot**

**7.5**

**8.0 Unbearably Hot**



# **BORG SCALE**

## **RATING OF PERCEIVED EXERTION**

**6**

**7 - Very, very light**

**8**

**9 - Very light**

**10**

**11 - Fairly light**

**12**

**13 - Moderately hard**

**14**

**15 - Hard**

**16**

**17 - Very hard**

**18**

**19 - Very, very hard**

**20**

## **PAIN INTENSITY**

**0 - No pain at all**

**½ - Very faint pain**

**1 - Weak pain**

**2 - Mild pain**

**3 - Moderate pain**

**4 - Somewhat strong pain**

**5 - Strong pain**

**6 -**

**7 - Very strong pain**

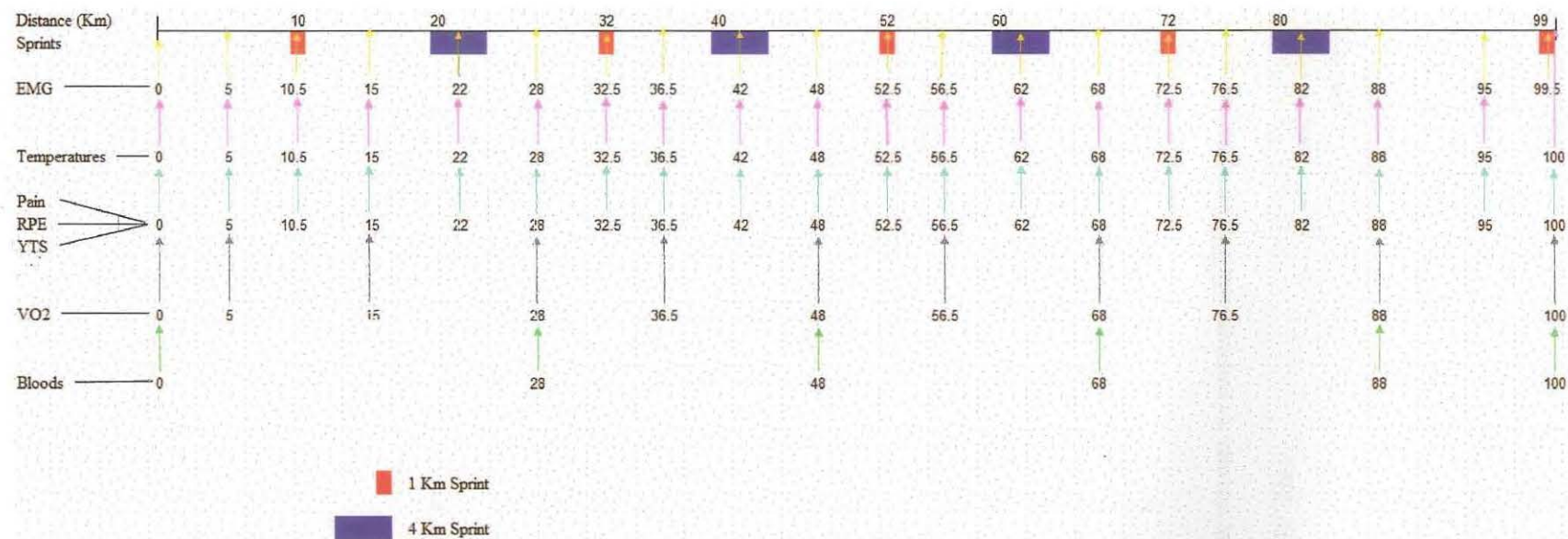
**8 -**

**9 -**

**10- Extremely intense pain**

**(almost unbearable)**

## EXPERIMENTAL PROTOCOL



MULTIDIMENSIONAL FATIGUE INVENTORY  
\*\*\* MFI-20 \*\*\*

Instructions:

By means of the following statements we would like to get an idea of how you have been feeling lately. There is, for example, the statement

"I FEEL RELAXED"

If you think that this is entirely true, that indeed you have been feeling relaxed lately, please, place an X in the extreme left box, like this

yes (entirely true) 

--	--	--	--	--

 no (not entirely true)

The more you disagree with the statement, the more you can place an X in the direction of "no" that is not true". Please, do not miss out a statement and place one X next to each statement

1. I feel fit	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
2. Physically: feel only able to do a little	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
3. I feel very active	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
4. I feel like doing all sorts of nice things	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
5. I feel tired	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
6. I think I do a lot in a day	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
7. When I am doing something, I can keep my thoughts on it	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
8. Physically: I can take on a lot	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					
9. I dread having to do things	yes (entirely true) <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td><td style="width: 20px; height: 20px;"></td></tr></table> no (not entirely true)					

10. I think I do very little in a day	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
11. I can concentrate well	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
12. I am rested	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
13. It takes a lot of effort to concentrate on things	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
14. Physically I feel I am in a bad condition	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
15. I have a lot of plans	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
16. I tire easily	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
17. I get little done	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
18. I don't feel like doing anything	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
19. My thoughts easily wander	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true
20. Physically I feel I am in an excellent condition	yes that is true	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>	no that is not true

Thank you very much for your cooperation